AIR RAID PRECAUTIONS
HANDBOOK No. 3

(1st Edition)

MEDICAL TREATMENT OF GAS CASUALTIES

LONDON
HIS MAJESTY'S STATIONERY OFFICE
Price 6*d.* net

AIR RAID PRECAUTIONS
HANDBOOK No. 3
(1st Edition)

MEDICAL TREATMENT OF GAS CASUALTIES

Issued by the Home Office
(Air Raid Precautions Department)

LONDON
PRINTED AND PUBLISHED BY HIS MAJESTY'S STATIONERY OFFICE
To be purchased directly from H.M. STATIONERY OFFICE at
the following addresses :
Adastral House, Kingsway, London, W.C.2 ; 120 George Street, Edinburgh 2 ;
26 York Street, Manchester 1 ; 1 St. Andrew's Crescent, Cardiff
80 Chichester Street, Belfast ;
or through any bookseller

1937

Price 6*d.* net

List of Air Raid Precautions Handbooks issued and projected

No. 1 Personal Protection against Gas (*1st edition*) price 6*d.* : 8*d.* post free.

A handbook primarily for members of air raid precautions services.

No. 2 First Aid for Gas Casualties (*2nd edition*) price 4*d.* : 5*d.* post free.

A handbook designed primarily for services giving first aid to air raid casualties.

No. 3 Medical Treatment of Gas Casualties (*1st edition*) price 6*d.* : 8*d.* post free.

A handbook for hospitals and doctors.

No. 4 Decontamination of Materials (*1st edition*) price 6*d.* : 7*d.* post free.

Decontamination of streets, buildings and their contents, vehicles and plant.

No. 5 Structural Precautions against Bombs and Gas (*in preparation*).

Structural protection against bombs and gas in buildings; air raid shelters, either separate or in buildings. This handbook will be designed for use by architects, builders, and others directly responsible for the construction or maintenance of buildings.

No. 6 Air Raid Precautions in Factories and Business Premises (*1st edition*) price 6*d.* : 7*d.* post free.

A handbook designed primarily for the guidance of occupiers of such premises.

No. 7 Anti-Gas Precautions for Merchant Shipping (*2nd edition*) price 3*d.* : 4*d.* post free.

Includes certain recommendations to Port Authorities.

A special Handbook is also being prepared for Householders.

In addition to these Handbooks, there is published a series of A.R.P. Memoranda dealing with various aspects of the organisation to be provided by local authorities for public air raid precautions services (see list on back cover).

GENERAL PREFACE

The series of Air Raid Precautions Handbooks (of which a list is given on the opposite page) is produced, under the authority of the Secretary of State, by the Air Raid Precautions Department of the Home Office with the assistance of other Government Departments concerned.

The measures for safeguarding the civil population against the effects of air attack which these Handbooks describe have become a necessary part of the defensive organisation of any country which is open to air attack. The need for them is not related to any belief that war is imminent. It arises from the fact that the risk of attack from the air, however remote it may be, is a risk that cannot be ignored, and because preparations to minimise the consequences of attack from the air cannot be improvised on the spur of the moment but must be made, if they are to be effective, in time of peace.

For the purpose of the measures now to be taken, it must be assumed that the scale of attack would greatly exceed anything which was experienced in the last war, and would involve the use of high explosive and incendiary bombs.

The use of poison gas in war is forbidden by the Geneva Gas Protocol of 1925, to which this country and all the most important countries of western Europe are parties, and the Government would use every endeavour on an outbreak of war to secure an undertaking from the enemy not to use poison gas. Nevertheless, the risk of poison gas being used remains a possibility and cannot be disregarded.

The Handbooks are designed to describe a scheme of precautions which it is hoped would prove effective in preventing avoidable injury and loss of life, or widespread dislocation of national activities. The Handbooks aim at giving the best available information on methods of passive defence against air attack, and will be revised from time to time in the light of future developments.

CONTENTS

Provisional issue, September, 1936 (7,000 copies).
1st edition, July, 1937.

BIBLIOGRAPHY

The following books on the treatment of gas casualties are also recommended.

(i) *Official publications* (*H.M. Stationery Office*)—

Official History of the War. Medical Services, Diseases of the War, Vol. II, price £1 5s. net.

Official History of the War. Medical Services, Surgery of the War, Vol. II, price £1 5s. net.

Manual of Treatment of Gas Casualties (1930), price 2s. net.

Defence against Gas (1935), price 1s. net.

R.A.M.C. Training (1935), price 3s. 6d. net.

(ii) *Non-official publications*—

Foulkes, Major-General C. H. "Gas", The Story of the Special Brigade. (Edinburgh and London, William Blackwood & Sons, Ltd., 1934), price 30s. net.

Mitchiner, Philip H. The Modern Treatment of Burns and Scalds. (London, Baillière, Tindall and Cox, 1935), price 5s. net.

Vedder, Edward B. The Medical Aspects of Chemical Warfare. (Baltimore, U.S.A., Williams and Wilkins, 1925), price $6.50 net.

Winternitz, M. C. Collected Studies in the Pathology of War Gas Poisoning. (Yale University Press, 1920), price 45s net.

INTRODUCTION

This handbook on the treatment of gas casualties has been written primarily for doctors. The material from which it is compiled is being used jointly for this book and for a similar book for Medical Officers of the Defence Services. The subject is treated here from the aspect of civil air raid precautions.

Study of the medical aspects of gas warfare has, since the last war, been confined to a small circle concerned with the protection of the Defence Services. The development of aircraft has now, however, made it clear that in a future war no part of these Islands might be wholly immune from air attack, and it is therefore impossible to neglect the protection of the civilian population also against the dangers and effects of gas. For this reason it is hoped that the medical problems to which poison gases give rise will come to be included as subjects of general study and research among the medical profession.

It is considered to be a matter of national importance that as many as possible of the medical practitioners in the country should learn the effects of war gases and the appropriate methods of treatment. This is necessary so that, in the event of air attack accompanied by the use of gas, they can treat casualties among the civilian population; and in addition they will be able in time of peace to speak with authority on occasions when reference is made to the dangers of gas in war. Great importance is attached to the doctor, with the influence of his professional status, having the knowledge and authority to speak on the problems which might arise if gas should unfortunately ever be used against our people. On this, as on other matters, a doctor should be prepared to be consulted by his patients.

This handbook deals with the clinical aspect of anti-gas precautions. Particular information on methods of personal protection against war gases is given in A.R.P. Handbook No. 1—Personal Protection against Gas. It is hoped that every doctor will read that handbook, without which knowledge of the medical treatment of gas casualties will be incomplete.

There is in addition a separate book—A.R.P. Handbook No. 2, First Aid for Gas Casualties—which has been specially written for first aid personnel and nurses. That book too is recommended to all medical practitioners.

CHAPTER I

GENERAL DESCRIPTION OF WAR GASES

1. Meaning of " Gas " in Chemical Warfare

The term " gas," in connection with warfare, is used in a very general sense, to include any chemical substance, whether solid, liquid or true gas, employed for its poisonous or irritant effects on the human body.

Such substances are, generally speaking, dispersed in the air as vapours or as poisonous smokes, and exert their action on persons breathing the air thus contaminated. Some of them, however, such as the blister gases, whether in the form of liquid or vapour, have also the power of acting directly on the skin.

Gases are generally classified in two main categories.

(*a*) *Non-persistent* substances which, when liberated, are rapidly converted into gas or smoke. Clouds of gas so produced continue to be effective only until dissipated by dilution with the surrounding air.

(*b*) *Persistent* substances, which are generally liquids. These liquids contaminate the objects with which they come in contact, and continue to give off vapour for a considerable period; mustard gas, lewisite and most tear gases are typical examples. Both the liquid and the vapour are poisonous.

2. Notes on the Use of Gas in the last War

Chemical warfare in the modern sense was first introduced by the Germans in April, 1915, on the Western Front, chlorine gas being used by them in successive attacks until May of the same year, when tear gases also were used. These attacks found the Allies not only unprepared, but also inexperienced in the effects that chlorine produces on its victims. The

casualties sustained during this period of ignorance and unpreparedness cannot be estimated accurately, but they were very heavy, and improvised means of protection had to be adopted at once.

The first official respirator (a cotton pad soaked in thiosulphate of soda, glycerine and sodium carbonate) was issued in May, 1915, and after that date defence, on the whole, kept ahead of attack—so much so that phosgene gas, first used by the Germans in December, 1915, found the Allies relatively well protected against its effects.

With a view to overcoming this protection the Germans introduced the arsenicals (or "nasal irritants") and mustard gas. The former were intended to penetrate the box respirator which was then in use by the Allies, while the latter, having a very faint smell and causing no immediate irritation, might be expected to take effect before the need for putting on a respirator was realised. Comparative failure attended the use of the arsenicals; mustard gas, on the other hand, was only too successful, for, in addition to its insidious latency, the gas possessed the power to attack all parts of the body not protected by the respirator and had, in addition, the great offensive value of persistency.

Much valuable information on the medical aspect of gas casualties is contained in the Official History of the War, Medical Services, "Diseases of the War," Vol. II.

The success secured by the use of mustard gas in the last war was chiefly due to the action of the vapour alone—vapour emitted from persistent deposits of the liquid Mustard gas was then only used in shells; but there is no practical difficulty in using it in bombs or in the form of spray discharged by aircraft, when contamination of the bare skin and of clothing by the liquid might cause casualties over wide areas, if no precautions were taken.

3. Objects to be achieved by the Use of Gas

The objects which an enemy might seek to achieve by the use of gas may be summarised as follows:—

(*a*) To produce casualties by means of the gas itself.

(*b*) To produce casualties by contaminating streets, etc.

(*c*) To reduce, or even to arrest, industrial and commercial activities.

(*d*) To make food and similar stores unfit for use. (A separate handbook is being prepared on the protection of food supplies.)

(*e*) By causing widespread discomfort, anxiety and disablement, to lower the morale of the civil population and induce a will to compromise or surrender.

The type of gas which might be used would naturally vary with the object in view.

The respirators which will be provided by the Government will give protection for the eyes, nose, face and lungs against any type of gas which, so far as is known, could be used in war.

In view of the properties of blister gases, however, which are capable of attacking other parts of the body which the respirator cannot protect, advice is being given to the public to remain indoors in a gas-protected room during an air raid. Essential air raid personnel who might have to be exposed to risk of contamination in performing public services will be supplied by the Government with the necessary protective clothing.

4. Factors governing the Use of Gas

It is evident that the higher the concentration of the gas, the shorter will be the period of exposure required to produce pathological damage; and the converse also holds good.

The effective use of gas may be markedly influenced by meteorological conditions as well as by topographical features in the area affected.

A strong wind will rapidly dilute and disperse all concentrations of non-persistent gases, while in the case of a persistent gas the rate of evaporation of the liquid will be increased, thus tending to clear the area more rapidly. With a low wind velocity, on the other hand, a high local concentration may be obtained with both types of gases, and the persistency of such liquids as mustard gas will be markedly lengthened, in the absence of other adverse factors.

Temperature plays an important rôle, both by influencing the diffusion of the gas by convection currents and by affecting the persistence of such liquids as mustard gas; hence warm sunny weather is inimical to the most effective use of gas. On the other hand, very cold weather has its disadvantages too, as it very markedly reduces the immediate value of persistent gases—in the case of mustard gas, for example, evaporation will be very greatly reduced as this liquid freezes at comparatively high winter temperatures. Mustard gas in the frozen state however is not by any means inert, as contact with it under these conditions will still produce a burn. If the frozen mustard gas contamination is carried (e.g. on boots) to warmer surroundings it will soon liberate an effective vapour concentration.

Conditions of excessive moisture are also unfavourable to the effective use of persistent gases, as rain tends slowly to destroy them or to wash them away.

The most favourable meteorological conditions for the employment of the two main types of gases are the following:—

Non-Persistent gases.

(*a*) A low wind velocity.

(*b*) Clear nights (cloudy days and nights are slightly less favourable, while clear sunny days are the least favourable).

Persistent gases.

(*a*) A low wind velocity.

(*b*) A high ground temperature.

(*c*) Absence of heavy rain.

Generally speaking, a clear, still night offers the most favourable conditions for the use of non-persistent gas. At such times the absence of air movements causes the gas to dissipate very slowly with the result that a high concentration is maintained for a long time.

In the case of persistent gases, a high ground temperature is usually the most important consideration, since this will induce rapid evaporation of the gas and the formation of a high local concentration of the vapour.

5. Medical Classification of Gases

The gases which, so far as can be anticipated, might be used in time of war may be classified as follows, the alternative names given in brackets being those by which the gases are described for purposes of civilian anti-gas training.

It is impossible to forecast which of these gases is likely to be chosen for chief use, but there seems a possibility that several different types might be employed simultaneously in order to confuse the measures for defence and treatment.

GROUP I. *Gases which may produce disablement or death.*

(a) **Vesicants** (or "Blister Gases"), such as **mustard gas** and **lewisite.** These are substances which, whether in the liquid, solid or vapour state, will damage any part of the body with which they come in contact. Typical effects of the vapour are acute conjunctivitis, inflammation of the mucous membrane lining the respiratory tract, and burning of the skin varying from erythema to vesication. The effect of the liquid on the skin is severe vesication.

When death occurs after the inhalation of mustard gas, it is usually from a complicating septic bronchitis with broncho-pneumonia, while as a result of very

extensive skin vesication death may result from secondary shock or sepsis.

(b) **Asphyxiants** (or " Lung Irritant Gases "). These gases, which include **chlorine, phosgene, di-phosgene** and **chloropicrin,** are essentially lung irritants exerting their main action on the pulmonary alveoli although the upper respiratory passages are affected in addition. They are used primarily as lethal agents, and, in the absence of an efficient respirator, their action usually results in a pulmonary oedema which may be fatal.

(c) **Paralysants,** such as **hydrocyanic acid** and **hydrogen sulphide.** These highly toxic gases were used but did not prove a success in the last war. More effective methods of liberating them may, however, be found and knowledge of them from the medical aspect is desirable. In high concentration, both these gases can produce death rapidly through paralysis of the respiratory centre.

GROUP II. *Gases used primarily as harassing agents.* (These gases may not produce casualties, but they cause temporary distress and compel the wearing of a respirator.)

(a) **Lachrymators** (or "Tear Gases"), such as **chloraceto-phenone (C.A.P.), ethyl-iodo-acetate (K.S.K.)** and **bromo-benzyl-cyanide (B.B.C.).** Even low concentrations of gas given off by these compounds will immediately irritate the eyes, causing profuse lachrymation and intense spasm of the eyelids—symptoms, however, which disappear on leaving the contaminated area. In very high concentrations they may act as acute lung irritants.

(b) **Nasal Irritants** (or " Nose Irritant Gases "). These are organic arsenical compounds such as **di-phenyl-chlor-arsine (D.A.), di-phenyl-amine-chlor-arsine (D.M.)** and **di-phenyl-cyano-arsine (D.C.).** These solid arsenicals, when suitably dispersed, produce clouds of minute particles which, if inhaled even in

low concentrations, will produce symptoms of acu physical distress. These symptoms are distressi gnawing pain in the nose and chest, with lachrym tion, salivation and even vomiting. Inferior respir tors are penetrated by these fine particles. T symptoms, however, are temporary, and, althou alarming at the time, usually subside within an ho after removal from the affected area.

GROUP III. *Gases liable to be encountered under w conditions.* (These gases are not likely to be us directly as chemical warfare agents in the ordina sense.)

(a) **Carbon monoxide.** This dangerous gas, thou not used offensively as a war gas, is frequently m with in the course of mining and tunnelling oper tions, in the interior of burning buildings, and genera wherever combustion occurs in the absence of adequate supply of oxygen. Typical instances occ in confined spaces following the burst of a high exp sive shell, or from the use of slow combustion stov charcoal braziers, or internal combustion engines such spaces. It is a constituent of ordinary illuminati gas, leakage of which may cause serious poisonin The gas produces its insidious effects through its w known interference with the respiratory functions the blood. Ordinary respirators give no protection.

(b) **Nitrous fumes.** These gases are given off burning cordite, or when detonation of nitro-exp sives is incomplete. They act as powerful and ve insidious lung irritants, with delayed symptoms sembling those of phosgene poisoning, and it is i portant to remember that they are often accompan by carbon monoxide. Respirators generally aff partial protection against nitrous fumes, but nc against the carbon monoxide.

(c) **Screening smokes.** Various chemicals may used to provide smoke screens for concealing import areas or buildings. Such substances as **phosphor**

hlorosulphonic acid, titanium-tetrachloride, and a umber of the **chlorinated-hydrocarbon series** are tilised for this object, while phosphorus may also be sed in bombs for incendiary purposes. These smokes re non-toxic in the open, but serious effects may follow he bursting of the bombs at close quarters.

(d) **Fumes which may be encountered in fire-ghting.** Apart from the risk of encountering carbon nonoxide, and possibly nitrous fumes, when fighting res in confined spaces in wartime, an additional danger nay arise through toxic gases evolved by fire-xtinguishing chemicals. Apart from its possible oxicity, such an atmosphere may be seriously eficient in oxygen.

The order in which these gases are described in the ubsequent chapters is that adopted in other A.R.P. Handbooks as being the most convenient for purposes f teaching.

A summary Table of Gases is given in Appendix A. ppendix B gives the formulae and physical constants f the gases.

CHAPTER II

MEDICAL ASPECTS OF CIVIL AIR RAID PRECAUTIONS

Before dealing in the following chapters with the medical aspects of the treatment of gas casualties, it is desirable to survey the position of civil air raid precautions as it especially affects a medical practitioner.

6. Need for Anti-Gas Training for Doctors

For his own protection in the event of gas attack by air, and to enable him to continue to look after his patients, even if he undertakes no other duties, every doctor should know how to wear a respirator, and what precautions are needed when gas is present. Information on these subjects is given in A.R.P. Handbook No. 1. (Personal Protection against Gas.) A course of anti-gas training, including practice in wearing a respirator, etc., is essential if a full knowledge is to be obtained. This training can be obtained from the medical instructors working under the Air Raid Precautions Department.

An understanding of the nature and use of respirators and protective clothing, such as may be obtained from A.R.P. Handbook No. 1, is necessary for a proper understanding of the chapters which follow.

7. Proposed Civilian Casualties Organisation

The organisation for dealing with civilians injured in air attack will be the responsiblity of the local authorities. It comprises a first aid organisation to be manned largely by the St. John Ambulance Brigade, the St. Andrew's Ambulance Association and the British Red Cross Society, supported by arrangements for hospital treatment.

The first aid organisation will consist of first aid parties, to give first aid to casualties on the spot; and first aid posts, at frequent intervals in urban areas, to deal with walking cases and the less seriously injured. The first aid posts will also include accommodation for washing and the changing of clothing in cases of persons, injured or uninjured, who have become contaminated with blister gas. An ambulance service will be provided in conjunction with the first aid organisation.

Hospitals will be organised in two classes—casualty clearing hospitals for the direct reception of the seriously injured (who should normally not be taken to a first aid post), and base hospitals in less dangerous areas to which the patients in casualty clearing hospitals could be evacuated at the earliest possible moment. In many cases the base hospitals would have to be taken over from other uses, or improvised, at the outbreak of war.

The details of this organisation are contained in A.R.P. Memorandum No. 1. (Organisation of Air Raid Casualties Services).

It will probably be impossible for doctors to accompany first aid parties or for one to be permanently on duty at every first aid post. Nevertheless doctors can in peace time be of great help in training the personnel of the St. John Ambulance Brigade, the St. Andrew's Ambulance Association and the British Red Cross Society. In time of war, doctors will be required to supplement the staffs of existing hospitals and to form the staffs of new base hospitals; and shortage of hospital beds may make it necessary to discharge less serious casualties to their homes before they have ceased to require medical attention.

Doctors who desire to offer their services for air raid casualty duties should approach the Medical Officer of Health of their local authority.

8. Decontamination of Clothing and Materials contaminated with Blister Gas

As will be made clearer in the following chapters, the blister gases leave on all kinds of material objects a contamination which will give rise to personal injury if any one comes into contact with them.

Clothing in particular will be dangerous to wear, when contaminated, until it is decontaminated. The processes of decontamination for clothing, and also for stretchers, are described in A.R.P. Handbook No. 1—Personal Protection against Gas. (See also Sections 31, 33 and 43 of this handbook.)

The decontamination of other material objects, which is an elaborate business, is dealt with in A.R.P. Handbook No. 4—Decontamination of Materials.

Important Note

No description of decontamination is given in this book because it is not a subject which can safely be summarised. It is desirable that every medical practitioner should be thoroughly conversant with the matter and should obtain detailed information from A.R.P. Handbooks Nos. 1 and 4.

CHAPTER III

LACHRYMATORS OR " TEAR GASES "

9. Characteristics and Description of Tear Gases

The lachrymators, and the nasal irritants which are described in the next chapter, possess certain important general features:—

(1) Their action is selective, i.e., they usually only attack exposed sensory nerve endings or mucous membranes, such as those of the eye, the naso-pharynx and the respiratory tract.

(2) They are effective in extremely low concentrations.

(3) Their effects are immediate, but temporary. Withdrawal from the gas-charged atmosphere is followed by rapid recovery. Permanent disabilities rarely occur, though such cases may be experienced after an unusually heavy dosage.

There are many compounds, both liquid and solid, which may be used as lachrymators in wartime. The following are typical examples:—

(*a*) *Chlor-aceto-phenone* (*C.A.P.*).—A colourless, crystalline solid melting at 54° to 59° C. (i.e., 129·2° to 138·2° F.) and boiling at 245° C. (473° F.). Though only sparingly soluble in water, it dissolves readily in all the organic solvents. Chlor-aceto-phenone is a very stable compound which does not decompose on heating or detonation; its lachrymatory effects, however, are soon lost by reason of the condensation of the substance to the solid, inert state soon after the initial dispersion, and it is therefore classed among the non-persistent gases. This gas is used in gas chambers or vans in the course of anti-gas training. It is also the gas used by the authorities in some foreign countries for dispersing mobs.

(*b*) *Ethyl-iodo-acetate* (*K.S.K.*).—A dark brown, oily liquid with a smell resembling that of " pear drops ". Its high boiling point (180° C. or 356° F.) and comparatively low vapour pressure ensure for it a certain degree of persistence on the ground.

(*c*) *Bromo-benzyl-cyanide* (*B.B.C.*).—In the pure state this is a yellowish, crystalline solid, stable at ordinary temperatures and melting at 24·8° C. (76·6° F.). In the crude form, as employed in war, B.B.C. is a heavy, oily, yellow liquid with a penetrating, bitter-sweet smell. The liquid boils at 242° C. (467·6° F.); it is more stable, and has a lower vapour pressure than ethyl-iodo-acetate, hence it persists longer than the latter as an effective lachrymator when spread on the ground.

10. Action of Lachrymators on the Body

Exposure to any of these lachrymators gives rise to an immediate, acute and localised irritation of the sensory nerve-endings on the corneal and conjunctival surfaces, which may vary from a mild irritation to an intense stinging sensation according to the strength of the concentration. Through reflex action, this is followed by profuse watering of the eyes and spasm of the eyelids, and the latter may be so acute as to render it impossible to keep the eyes open.

With a rise in the concentration of the vapour, further effects may make their appearance. The irritant action of the gas on the respiratory passages and lungs produces a burning feeling in the throat and discomfort in the chest, and, if the exposure be continued, nausea and vomiting may result.

C.A.P. possesses the characteristic attribute, absent in the case of the liquid lachrymators, of irritating the bare skin, especially if the skin be hot and moist. This irritant action is not sufficiently serious to necessitate special protective garments. Persons with dry skins,

however, who do not sweat, are inclined to be hypersensitive to C.A.P. and may develop an obstinate dermatitis if often exposed to the vapour.

As a rule, persons exposed to lachrymators never exhibit more severe symptoms than those described above, as the very high concentrations necessary to produce lung lesions are not met with in the open. In confined spaces, however, where accidental splashes of the actual liquid on the skin may occur, or the inhalation of a high concentration may be experienced, more severe results may be expected: these vary from severe conjunctivitis, with tracheitis and bronchitis from the effects of the vapour, to blistering of the skin, keratitis and corneal opacities after contamination with the liquid.

The rare fatalities that occurred in the last war were characterised by a severe pulmonary oedema following the accidental inhalation of massive concentrations of the gas.

The respirator affords complete protection to both eyes and lungs against all concentrations of tear gases likely to be met in war. The use of goggles alone is not recommended, as, in addition to the liability of leakage and constant dimming, they offer no protection to the respiratory tract.

For practical reasons it is important to recognise the difference between the effects produced on the eyes by " tear gases " and by " blister gases." In the case of tear gases the person affected is for the moment blinded by his own tears and by his natural instinct to close his eyes to minimise the irritation: he may require assistance to get away from the poisonous air, but that is all, for in pure air the effects will quickly pass away. In the case of a " blister gas " like mustard gas the inflammation comes on slowly, but when once it has become established it lasts for some time, and perhaps for a day

or two the sufferer will be quite unable to see owing to the swelling of the eyelids and the inflammation of the surface of the eye.

11. Treatment for Lachrymators

In the great majority of cases, adjustment of the respirator will suffice to alleviate the symptoms, and usually to clear up the condition completely. Experience has shown that even after severe exposures all symptoms disappear within 12 hours. No treatment, other than simple symptomatic, is usually necessary, and no after-effects follow exposure. In the rare cases where acute conjunctivitis or respiratory affections develop, treatment should be symptomatic and follow general principles. If the eyes be contaminated by the actual liquid from gas sprays, bursting bombs, etc., lavage with normal saline will be necessary.

CHAPTER IV

NASAL IRRITANTS OR "NOSE IRRITANT GASES"

12. Characteristics and Description of Nose Irritant Gases

These sensory irritants are solid arsenical compounds which can be dispersed by heat or detonation in the form of a very fine, almost invisible, particulate cloud or smoke. They share with the lachrymators the general characteristics described at the beginning of Section 9 in the previous chapter.

The object underlying their original employment in the last war was to penetrate the respirator container in use at that time, in the belief that the distressing symptoms, following even a brief exposure, would induce men to discard their respirators as useless; this would expose them to the effects of lethal gases, such as phosgene, which were often released simultaneously. The nasal irritants did not meet with any striking success, because the methods in use at that time for the release of the gas were unsatisfactory.

The following are typical examples of the nasal irritants:—

(*a*) *Di-phenyl-chlor-arsine* (*D.A.*):—A colourless, crystalline solid melting at 38° to 40° C. (100·4° to 104° F.) and boiling at 333° C. (631·4° F.) at ordinary atmospheric pressure; insoluble in water.

(*b*) *Di-phenyl-amine-chlor-arsine* (*D.M.*):—A yellow, almost odourless, crystalline solid melting at 195° C. (473° F.) and boiling (with decomposition of the compound) at 410° C. (770° F.) at ordinary atmospheric pressure; insoluble in water, and difficult to disolve in the ordinary organic solvents.

(*c*) *Di-phenyl-cyano-arsine* (*D.C.*):—A colourless, crystalline solid, almost entirely odourless, with a melting point of 33° C. (91·4° F.) and boiling at 346° C. (654·8° F.) at ordinary atmospheric pressure; almost insoluble in water, but dissolving readily in oils and in organic solvents.

These compounds are solids at ordinary temperatures, but when heated or dispersed by an explosive they are vaporised, without decomposition, in the form of an almost invisible cloud of minute particles which remain suspended in the air.

13. Action of Nose Irritant Gases on the Body

The main feature of these arsenical irritants is their power of causing violent sensory irritation in man even though present in extremely low concentrations.

The effectiveness of the arsenical gases depends on the amount inhaled before symptoms (which are delayed in onset for some minutes) make themselves felt. Even if the affected person withdraws from the poisonous atmosphere directly the irritant effects are felt, the symptoms continue to increase in severity for some time before they begin to subside. Unlike the tear gases, therefore, whose sensory effects are immediate but disappear rapidly on adjustment of the respirator, the arsenical irritants are characterised by an increase in the severity of the initial symptoms after adjusting the respirator or leaving the gas-laden atmosphere. This relatively slowly developing action of the arsenicals and the subsequent aggravation of symptoms constitute their chief danger in the case of uninformed persons, as they may engender distrust in the respirator.

The symptoms are characteristic, and consist of acute pain in the nose and accessory sinuses with a sense of " fullness " in the head and repeated sneezing (hence the term " sternutators " often applied to

these gases). A burning sensation in the throat, and one of tightness and pain in the chest, also a feeling of grittiness in the eyes with pain and lachrymation, and aching of the gums, are common, while salivation, with nausea or even vomiting, are important symptoms. Acute mental distress is very marked in severe cases, who feel and look utterly miserable. This condition of intense discomfort is very alarming to the inexperienced.

The irritant effects are transitory, and symptoms usually disappear within an hour. Even in severe cases no lasting organic lesion is likely to follow exposure to these gases in the open.

The respirator charcoal, which arrests the lethal gases, has little or no protective value against the particulate clouds of these arsenical compounds, but special filtering devices are added to trap these particles or smokes.

Symptoms of true arsenical poisoning may occur through persons using water drawn from bomb or shell craters contaminated with these arsenicals. The arsenic content in these craters may be very high, and men have been known to suffer from dermatitis after shaving with water drawn from them.

14. Treatment for Nose Irritant Gases

In the great majority of cases a brief period of rest is the most that is required. In a few exceptionally severe cases, however, pain may call for medical relief, when the inhalation of a little chloroform may be found of transitory assistance. A 5 per cent. solution of sodium bicarbonate for nasal irrigation or as a gargle will help to allay the irritation of the nose and throat.

If a respirator is being worn, care should be taken to remove the clothes previous to taking off the respirator, or the arsenical particles shaken out of the clothes may be inhaled.

CHAPTER V

ASPHYXIANTS OR "LUNG IRRITANT GASES"

15. General Description of Lung Irritant Gases

The chief members of this group are chlorine, phosgene, di-phosgene and chloropicrin. Chlorine and phosgene are true gases under normal conditions of temperature and pressure, while di-phosgene and chloropicrin are liquids of comparatively high boiling points—a quality which renders them somewhat persistent.

Chlorine was the gas first used by the Germans in the last war. On release from the cylinder, bomb, etc., it forms a greenish yellow cloud with a pronounced smell of bleaching powder. It is highly irritant to the mucous membranes of the upper respiratory passages, and a marked feature of its action is the violent and paroxysmal cough which it induces, and which persists even after the cessation of exposure.

Phosgene is the most important member of the group, and the most toxic. This gas is a liquid which boils at 8·2° C. (46·8° F.) with the evolution of a dense, colourless vapour. Although the least immediately irritant of the group, phosgene is readily detectable, even in concentrations which are harmless to the lungs, by its characteristic smell of musty hay.

Di-phosgene is an oily liquid boiling at 128° C. (262·4° F.) and smelling like phosgene. Apart from its more pronounced lachrymatory power, the symptoms produced by this gas are practically identical with those caused by phosgene.

Chloropicrin is a yellow liquid boiling at 112° C. (233·6° F.); its smell resembles that of chlorine. Chloropicrin is the most irritant member of the group, causing more sensory irritation of the respiratory passages than chlorine. It is also cumulative in its

action, and frequent exposures to small doses may gradually lead to a greatly increased susceptibility with a liability to attacks of nocturnal " asthma." It is also a strong lachrymator.

In view of the comparatively low toxicity of chlorine and chloropicrin when compared with phosgene, and also because of the ease with which their presence, even in low concentrations, is advertised by their immediate irritancy, it is less likely that either of these gases will be widely used in modern warfare in the field. There is no doubt, however, that they are dangerous gases if liberated in confined or built-up areas, and their moral effect if used against civilian centres might well be great.

16. Methods of Dispersion of Lung Irritant Gases

With the exception of chlorine, these asphyxiant gases may be dispersed by any type of projectile in ordinary use—shell, aircraft bomb, mortar bomb or Livens drum. In addition, both chlorine and phosgene may be dispersed from cylinders either mounted in fixed positions or carried on moving vehicles; in the case of phosgene the emission of the gas is hastened, in cold weather, by mixture with chlorine.

In the last war a combination of two or more gases of this group was almost invariably used in any cloud gas attack or gas shell bombardment. It appears doubtful, however, whether any asphyxiant gas other than phosgene would be employed in future wars, and, in view of the high concentration necessary to produce effective results, it is likely that phosgene will only be used in large calibre projectiles or in cylinders mounted on moving vehicles.

By whatever means these chemical agents are distributed, the resultant gas clouds are carried down wind. Action on unprotected persons in the path of the cloud is only limited by the period of their

exposure in the cloud. In the case of those who have respirators available the effective exposure is only the brief time occupied in adjusting the respirator.

Nevertheless, it must be borne in mind that the sudden release of a heavy cloud of asphyxiant gas in enclosed areas or in close proximity to personnel is fraught with danger even to persons who have (but are not wearing) respirators, since the intense spasm induced by a single breath of a heavy concentration of the gas may interfere with the quick adjustment of the respirator.

17. Mode of Action of Lung Irritant Gases

All lung irritant gases cause essentially the same type of pathological effect, this being most pronounced on the alveoli of the lungs and on the smaller bronchial tubes, and the great danger to be feared is the onset of acute pulmonary oedema. The rate of onset and the degree of oedema depend on the particular gas and on its concentration, and also, to a lesser degree, on the duration of the exposure.

These gases are also quite effective lachrymators, especially di-phosgene and chloropicrin; but they are far less powerful in this respect than the true lachrymators.

Their relative toxicity varies, except in the case of phosgene and di-phosgene where it is approximately the same. In lethal concentrations it is found that chloropicrin is about four times, and phosgene (or di-phosgene) practically ten times, more toxic than chlorine; and, while chlorine and chloropicrin cause more damage to the lining membranes of the respiratory passages, phosgene is more effective in the production of pulmonary oedema.

There is, however, little essential difference between the actions of the various members; the treatment and the prognosis for all alike depend on the extent of the lesions produced in the lungs. The extent of these

lesions varies as much with the concentration of the gas as with its particular nature, and they are to be determined by a consideration of the clinical features rather than by reference to the exact nature of the toxic substance.

As a type of the group, the morbid anatomy and the signs and symptoms of phosgene gas poisoning will now be described; differences from those of poisoning by other gases of the group will be considered as they arise.

18. Morbid Anatomy of Lung Irritant Cases

The essential lesions are pulmonary oedema, rupture of the pulmonary alveoli and concentration of blood, with increased viscosity and a tendency to thrombosis.

The earlier death ensues, the greater is the degree of pulmonary oedema found at *post-mortem* examination. Oedema may be fully established within two hours of gassing, when the lungs are found to be small or normal in size, heavy and completely waterlogged and with no sign of emphysema.

When death occurs later on the first day the lungs are voluminous, heavily oedematous and congested with blood, while aerated patches of emphysema, especially at the edges of the lungs, alternate with patches of collapse. On section, frothy serous fluid mingled with blood drips from the lung tissue, and petechial haemorrhages may be visible on the surface of the lungs. The pleural cavity almost invariably contains a quantity of serous, perhaps blood-stained, effusion which may vary in amount from two to twenty ounces.

In the case of deaths occurring on the second or third day, evidence of aeration of the lungs is present, especially in the lower lobes, and serous fluid does not drip so freely from the cut surface. With later deaths this dripping of serous fluids has ceased, and commencing broncho-pneumonia and pleurisy may indicate that secondary bacterial infection has set in.

The greater aeration of the lungs of cases dying on the second and third days, taken in conjunction with the clinical history of the severe cases that survive, makes it evident that the oedema fluid is rapidly absorbed from the lungs from the second day onwards.

In severe cases the haemoglobin percentages may rise as high as 140 with a corresponding rise in the red cell count. Associated with this concentration is the occurrence of thrombosis in the pulmonary blood-vessels, and also, to a variable extent, in those of other organs of the body.

Phosgene has relatively little effect on the upper air passages, but in chlorine and chloropicrin poisoning the bronchial tubes, and even the trachea, may show serious damage. The epithelial lining may be severely affected and desquamation may take place; the liability to blockage of these channels is therefore great, and the paroxysms of violent coughing, so typical of chlorine and chloropicrin poisoning, tend to induce a disruptive emphysema which is much more marked in these cases than in phosgene poisoning. Subcutaneous emphysema was rarely seen in the last war except after gassing by chlorine.

19. General Symptoms and Signs of Lung Irritant Poisoning

Exposure to an atmosphere containing phosgene or other pulmonary irritants may lead to two types of acute casualties:

(*a*) *Acute, with Violent Onset.*

Typical cases of this type are best seen after exposure to chlorine or chloropicrin; but even phosgene, in an effective concentration, causes immediate sensory irritation of the respiratory passages and catching of the breath, accompanied by a cough and a sensation of constriction and pain in the chest; this is followed by gasping respirations which are interrupted by fits of

violent coughing. Even after leaving the poisoned atmosphere the respiration remains rapid and shallow, and any attempt to draw a deep breath gives rise to painful discomfort and cough. Retching and vomiting are prominent features in the early stages of poisoning, and headache and a profound sense of fatigue often prostrate the patient.

In chlorine and chloropicrin poisoning a marked feature is the paroxysmal cough, accompanied by vomiting, which not only occurs during the exposure, but persists for a long time afterwards.

With the onset of oedema the breathing becomes rapid and panting, but of a characteristically shallow type; the ears and lips, and eventually the whole face, assume a cyanotic tint which, in cases of chlorine poisoning, may deepen to the intense violet of fullest cyanosis, with visible distension of the superficial veins of the face and neck.

In phosgene poisoning this full cyanosis is often omitted, and the patient passes rapidly into a state of circulatory collapse with a feeble, flickering pulse of over 120, a cold, clammy skin and a leaden hue of the face. In both these types of asphyxia the onset of cyanosis is marked by increasing restlessness and apprehension as the patient realises that he is being suffocated by his own secretions.

Apart from the fulminating cases in which death follows within two or three hours after exposure, lung irritant casualties can be divided into three types:—

(1) The milder case, with flushed face, somewhat rapid respiration and a painful cough.

(2) The severe case, with " blue " cyanosed face, distended neck veins and a full strong pulse of 100.

(3) The severely collapsed case, with a leaden " grey " cyanosis of the face and a rapid, thready pulse.

The mild case is often drowsy, and soon falls into a sleep from which he wakes refreshed. Coughing and a sense of debility may persist for a few days, and a temporary bradycardia may develop, but convalescence is assured.

Even cases of severe cyanosis tend to recover in two or three days if the colour is well maintained and the pulse rate does not exceed 100. At any time, however, and particularly if subjected to physical effort, these cases may rapidly pass into the dangerous condition of "grey" cyanosis and collapse, with thready and irregular pulse, from which they may not recover.

The mortality among the "grey" cases is high, death being due to failure of circulation, or to a fatal broncho-pneumonia which may interfere with recovery. When this infective complication develops, the sputum becomes purulent, the temperature rises and death usually follows rapidly.

(*b*) *Acute, with Insidious Onset.*

Cases occurred in the last war in which men who had been exposed to phosgene gas had been able to carry on their work for an hour or two with only trivial discomfort, and even to march from the trenches to their billets; thereafter they became rapidly worse and passed into a condition of collapse with a progressive pulmonary oedema which sometimes proved rapidly fatal.

In other cases men whose only complaint was a slight cough and tightness of chest had suddenly collapsed, and even died abruptly, some hours later on attempting to perform vigorous muscular work. Varying degrees of the same delayed effect of phosgene poisoning were met with, but in all cases the deficiency of oxygen (the result probably of pulmonary oedema already existing) had not been felt until muscular exertion increased the need for oxygen.

This delay in the onset of serious symptoms is not evident in chlorine gas poisoning, where the violent paroxysms of coughing, the painful dyspnoea, and the repeated attacks of vomiting convey the impression that the case is seriously ill from the start.

20. Physical Signs in Lung Irritant Poisoning

The percussion note may remain resonant over the chest, notwithstanding the existence of pulmonary oedema. The breath sounds are weakened, especially behind; they may also be harsh in character, but never tubular. Fine râles are heard, chiefly in the axillary region and at the back and sides of the chest, while rhonchi may be noted occasionally.

In the early acute stage the physical signs give little indication of the gravity of the case or the extent of the damage to the lungs. The colour, the pulse and the character of the respiration are the chief guides to prognosis. With the development of inflammatory complications and rising temperature the physical signs become those of pleurisy, bronchitis or broncho-pneumonia.

21. Prognosis of Lung Irritant Cases

Cases of the " blue " type which react favourably to the administration of oxygen usually do well, and if the circulation and the activity of the respiratory centre can be maintained the oedematous fluid in the lungs is absorbed within four or five days.

In the " grey " type, when cardiac weakness is increasing, the prognosis is bad; if recovery takes place it is often succeeded by a broncho-pneumonia which usually proves fatal. If, however, a case lasts into the third week after gassing, he may justly be expected to survive the acute infection. In 80 per cent. of the deaths due to phosgene or chlorine in the last war, the death occurred within 24 hours of gassing.

In the last war broncho-pneumonia was found to be more frequent and serious in men who had been suffering from bronchitis previous to gassing; similarly, men with pre-existing emphysema or lung disease were handicapped in their struggle against pulmonary oedema, since the margin available for respiration was correspondingly less.

22. Treatment of Lung Irritant Cases

Cases of all degrees of severity may be met with, and it may be difficult at times to decide whether or not a man has really been gassed. The benefit of the doubt should be given to the patient in all cases, and any man showing any feature of lung irritant poisoning should be rested for 24 hours for observation. It should be borne in mind that a delayed action may be exhibited by some pulmonary irritants, notably phosgene and the nitrous gases; but if no objective symptoms have arisen after the lapse of 48 hours the patient can be discharged.

(*a*) *Treatment in the Acute Stage.*

The essentials of treatment for acute poisoning by any pulmonary irritant gas are rest, warmth, venesection and oxygen. It is necessary to think of lungs choked by inflammatory oedema which none the less may be absorbed in three or four days if the circulation can be maintained for so long.

(1) *Rest.*—The importance of rest cannot be exaggerated; in the earlier stages undue muscular exertion may lead to an aggravation of the symptoms, while in the later stages, when the oxygen supply of the body is interfered with by pulmonary oedema, any attempt to perform muscular work may have disastrous consequences.

All cases should be treated as stretcher cases, and their clothing eased so as not to impede breathing. If this is not possible, walking cases should be given every assistance, so that they may avoid physical

effort as much as possible. As with every type of gas casualty, the patient's contaminated clothing should be removed on reaching the first aid post or hospital. Those who show definite symptoms should not be allowed to leave their beds or stretchers for any purpose whatever.

(2) *Warmth.*—This helps to combat shock, and also to diminish the oxygen consumption that is entailed by the muscular movements of shivering; attention should therefore be directed to this point when the patient's clothing is removed.

(3) *Venesection.*—As soon as cyanosis begins to appear, 15 to 20 ounces (300 to 600 ccs.) of blood should be withdrawn from a vein by a large bore needle. This treatment is beneficial for all cases except those of the leaden grey hue with failing circulation and a rapid, thready pulse. For this latter group it is harmful. After venesection the headache often disappears, dyspnoea is somewhat diminished and sleep usually follows. If these results follow, nothing further may be needed. But if symptoms of cyanosis and pulmonary oedema seem to be increasing, oxygen treatment should be begun at once.

Experiments carried out on animals late in the last war indicated that when venesection was combined with the intravenous infusion of isotonic salt solution still better survival results might be expected. There was no evidence that the infusion, when performed some time after venesection, led to an increase of lung oedema. Although this combined treatment was not applied to human casualties, it is possible that the method may prove a useful means of treating cases with pulmonary oedema when the haemoglobin estimation shows an unduly high concentration of the blood.

(4) *Oxygen.*—Oxygen should always be given to casualties with serious pulmonary oedema, that is, to those with intense blue cyanosis or grey pallor,

The aim should be to tide the patients over the critical period of the first two or three days, and for this purpose oxygen should be administered continuously by means of some special apparatus, such as Haldane's mask or a nasal catheter, that will ensure a suitable mixture with air.

The oxygen need not be warmed, and a sufficient current of it should be used (from two to ten litres a minute) to ensure a change in the patient's colour from livid blue or grey to a pink tint. If the light is insufficient to allow cyanosis to be judged, guidance may be obtained from the behaviour of the pulse. This treatment must be maintained, day and night if necessary, with a progressive lessening of the oxygen supply, until the patient does not lapse into cyanosis when the oxygen is withdrawn. When oxygen is given continuously over long periods, intermission should be made for five minutes every half hour.

If the supply permits, oxygen should also be given to the milder cases of oedema in order to prevent their lapsing into a more serious state of asphyxia.

The experience of the last war showed that no patient in whom it was possible to restore a pink colour by the proper use of oxygen died from simple pulmonary oedema. In a hospital likely to receive any considerable number of cases of lung irritant poisoning (not of broncho-pneumonia from mustard gas), it is helpful to establish for their treatment some definite routine of oxygen administration on the lines of the suggestions contained in Appendix C.

(*b*) *General Treatment.*

Serious cases are best treated in a well lit and well ventilated ward, protected from chill. The diet should be fluid and sparingly given in the acute stage, but bland drinks should be allowed freely.

Expectoration should be encouraged by some postural device; vomiting is helpful in emptying the lungs, and often occurs spontaneously, but it is liable to produce exhaustion, and it should not be induced by powerful drugs such as apomorphine or ipecacuanha. Raising the foot of the bed or stretcher three or four feet for a few minutes at a time, with the idea of draining fluid from the chest, is sometimes effective in helping free expectoration.

Expectorants should not be given to severe cases during the first two or three days for fear of increasing the tendency to cough and so augmenting the damage in the lungs. In mild cases, or when the acute symptoms have abated in the severe cases, ordinary expectorant mixtures containing ammonium carbonate and vinum ipecacuanhae may be given with advantage and are helpful in checking the possible development of infective bronchitis.

No drugs were found to be of any special value in the last war. Atropine did not prove effective in checking oedema or in relieving bronchial spasm, while morphia is dangerous and should only be used in small doses (1/16th gr.) to control extreme restlessness. The relief of asphyxia is the best means of relieving the headache and the best cardiac stimulant is oxygen.

If pulmonary complications develop (such as infective bronchitis, broncho-pneumonia, etc.), the patient should, if possible, be treated in a separate ward; otherwise, he should be separated by at least six feet from his nearest neighbour.

(*c*) *Treatment in the Convalescent Stage.*

No case should be moved, for purpose of convalescence, until definite cyanosis or severe symptoms have disappeared; it is also very important that a note of the special symptoms attending the acute illness should be forwarded with each case in order that subsequent treatment may be rightly controlled.

The milder casualties are likely to recover after a short rest; those who have passed through a stage of severe cyanosis, however, or who have suffered from a complicating broncho-pneumonia require a prolonged period of convalescence.

All except the more severe cases should be got up from bed as soon as possible; slight bronchitis or gastric disturbances, which usually are only temporary, do not contraindicate this, but cases of abnormally rapid or slow pulse should be rested a little longer.

A system of carefully graduated exercises, with full opportunities for lying down and resting in the intervals, should be instituted; the response to exercise of each individual, however, must be carefully studied, and exhaustion must be guarded against as symptoms of disordered action of the heart may develop, and add weeks or months to the period of convalescence.

23. After-effects of Poisoning by Lung Irritant Gases

Apart from infective broncho-pneumonia, which usually appears towards the end of a week and which may, in severe cases, develop the usual septic complications, the sequelae of poisoning by the asphyxiant gases are, contrary to popular belief, much less grave than was anticipated in the last war. The great majority of cases were restored to good health. Some continued to show inability for severe muscular effort or even for moderate exercise, associated with tachycardia and a rapid, shallow type of breathing. Recurring frontal headache, generally worse after exercise, and epigastric pain of a temporary nature were frequent, and, while pain in the chest was variable, the presence of a mild bronchitis was found in an appreciable proportion of cases.

In the last war, the two important, though not common, sequelae which tended to prolong invalidism were " D.A.H." and nocturnal " asthma."

" D.A.H." or irritable heart, with precordial pain, a sense of exhaustion, dyspnoea and persistent tachycardia after exercise but no evidence of organic heart disease, was the commonest and most persistent after-effect. A small proportion proved intractable, and there was evidence to show that this invalidism was increased if the men were pressed to physical effort too early and too fast at the beginning of convalescence.

Nocturnal asthma, which differed from the ordinary asthma of civil life, took the form of spasmodic attacks, lasting from three to thirty minutes, with shallow and rapid, but not difficult, respirations and with no abnormal physical signs in the chest during the attack. The pulse might be slow and full, or rapid and almost impalpable, and both the haemoglobin percentage and the red cells were increased.

Both disabilities almost always yield in time to a slowly progressive routine of graduated exercise, coupled with careful supervision and feeding, and firm reassurance to dispel neurasthenic factors.

Patients who have passed through a phase of infective broncho-pneumonia should always be considered as a group apart.

24. Invalidism after Lung Irritant Poisoning

Until knowledge came through clinical experience, there was much apprehension in the last war of permanent damage to the lungs after the inflammation caused by these chemical irritants. It was thought that severe emphysema and fibrosis of the lungs, or perhaps pulmonary tuberculosis, might result among the survivors. Nothing so gloomy occurred The after-histories of selected groups of cases were followed out in detail during the war, and the records of the Ministry of Pensions were analysed up to 1920 in order to produce the evidence related in the Official History of the War. Subsequent experience has

revealed nothing to alter the conclusions then reached. It was evident that men who had suffered from the most severe cyanosis with acute pulmonary oedema could recover rapidly and completely. Many such went back to full military duty after a convalescent period of from three to four months. Others, as described above, suffered from neurasthenic features of exhaustion or from temporary loss of wind and endurance. But a small proportion did develop permanent disability, with progressive dyspnoea, recurrent bronchitis and a radiographic picture of scattered fibrosis and emphysema in the lungs. Since it was proved that a man could recover completely from the effects of the chemical irritant, it is probable that these rare examples of chronic invalidism were due to slow fibrosis caused by the secondary complications of broncho-pneumonia.

As regards tuberculosis, a general survey of the situation in the last war was made, and it soon became evident that, in spite of what was being written in certain countries, there was, as would seem logical, no ascertainable connection between gassing and tuberculosis. On the other hand, it is unquestionable that gas aggravated any pre-existing tuberculous condition, where such was present, as would be expected.

CHAPTER VI

MUSTARD GAS

25. General Description of Mustard Gas

Mustard gas or di-chloro-di-ethyl sulphide (the " Yellow Cross " of the Germans and " Yperite " of the French) was certainly the most effective chemical agent used in the last war.

Lewisite, which is described in the next chapter, has similar properties.

Mustard gas, being a liquid with very persistent properties, may be discharged from the air either in bombs or as spray. Mustard gas bombs will disperse splashes of the liquid, larger near the crater and diminishing in size in the farther parts of the area affected, and these splashes will continue to give off dangerous vapour for days or even weeks unless the contamination is removed or neutralised. When discharged as spray it will fall in drops which will be larger or smaller according as the aircraft is flying low or high.

26. Physical and Chemical Properties of Mustard Gas

A knowledge of the physical and chemical properties of mustard gas is essential in order to understand its insidious action on the human body. Its outstanding characteristics are:—

Appearance.—In the pure state mustard gas is a clear, almost colourless, heavy and somewhat oily fluid with a faint mustard-like odour. In the crude form it is a heavy, dark coloured, oily fluid with a slightly more offensive smell resembling that of garlic or onions.

Odour.—In the absence of chemical methods for the ready detection of mustard gas, the sense of smell is the most reliable guide to its presence. The mustard-like or garlicky odour, though faint in low concentrations, is characteristic of the gas, and it is most

important that the smell should be memorised as part of anti-gas training. It is, however, well to remember that mustard gas may produce casualties in concentrations the smell of which may readily escape notice; also, that the sense of smell tires quickly, and that after a few minutes in a mustard gas atmosphere the smell of the gas may seem to have quite disappeared.

Boiling Point and Vapour Pressure.—The boiling point of mustard gas (217° C. or 422·6° F.) is high, and its vapour pressure is correspondingly low (0·05 mm.Hg. at 10° C. or 50° F., and 0·45 mm.Hg. at 40° C. or 104° F.)—hence its slow vaporisation at ordinary temperatures and its consequent quality of persistence.

Freezing Point.—The freezing point of pure mustard gas is 14·4° C. (57·9° F.), while that of the crude variety is considerably lower, viz., about 6° C. (42·8° F.)—somewhat high freezing points which limit the usefulness of the gas in cold weather, although contact with the frozen material is still a source of danger.

Density.—Mustard gas has a high specific gravity (1·28 at 15° C. or 59° F.) and, as it is not miscible with water, it readily sinks to the bottom when mixed with it.

Solubility.—Although mustard gas is only very slightly soluble in water (under 1 per cent.), both the liquid and the vapour are freely soluble in animal oils, and it is due to this lipoid solubility that mustard gas finds an easy entry into the skin. Other substances that readily dissolve mustard gas are alcohol, ether, petrol, benzene and kerosene or paraffin, carbon tetrachloride, acetone, carbon bisulphide, and many other organic solvents.

Stability.—Both physically and chemically mustard gas is a stable substance; it is unaffected by wide ranges of atmospheric temperature, though simple heat disperses it by hastening evaporation. It is only very

slowly hydrolysed by water; boiling water, however, hastens this decomposition, the products of which (hydrochloric acid and thiodiglycol) under ordinary circumstances are practically harmless. For its chemical neutralisation strong reagents are usually required, such as chlorine (as in bleaching powder), strong nitric acid, or other strong oxidising agents.

Penetration.—One of the most important physical characteristics of mustard gas is its power of soaking into most materials other than metals, glass, glazed articles, etc. This property, combined with its persistence, greatly complicates the problem both of defence and of decontamination.

27. Toxic Properties of Mustard Gas

Toxicity.—Mustard gas is an extremely dangerous substance both in the liquid and in the vapour state, but its action is essentially local, and no general systemic disturbance usually supervenes in the absence of secondary infection. The gas is not selective in its action, and any part of the body exposed to it is liable to suffer.

Vapour concentrations.—Atmospheres which contain low concentrations of mustard gas are particularly dangerous, as the comparative absence of smell in such concentrations renders them particularly insidious. The presence of the poison may escape detection and thus cause the exposure to be unduly prolonged.

Insidious characteristics.—The fact that there is no immediate irritation of the skin on contact with the liquid, nor of the eyes or respiratory tract on entering moderate concentrations of the vapour, constitutes one of the more serious dangers of this gas, as contamination may be unsuspected. Even when the gas has been detected by its characteristic odour, the sense of smell is soon dulled and the odour will cease to

be appreciated. If however the respirator is speedily adjusted the odour will be detected whenever the respirator is removed. It is important to remember, also, that harmful concentrations of the gas can easily be masked by innocuous smokes or other smells. in which case the gas will exert its effects undetected.

Delayed action.—After exposure to mustard gas vapour or contact with the liquid itself no effects are noticed for some time. Signs and symptoms do not begin to appear until after the lapse of two or three hours. Longer delay, even up to 24 or 48 hours, sometimes occurs.

Delayed healing.—It has already been stated that the action of mustard gas is local; the tissues affected are devitalised, they are easily injured by rubbing or pressure, and they are very prone to secondary infection. Where the gas has penetrated deeply, the healing process, even though sepsis be excluded, is very slow owing to damage to capillaries, veins and lymphatics. It is only when the action of the gas is superficial and localised that the condition clears up rapidly.

Sensitivity.—All persons are sensitive to the action of mustard gas, and so far as is known all who have not previously been exposed to its effects possess approximately the same degree of sensitivity irrespective of race or colour.

Acquired hypersensitivity.—In contrast to normal sensitivity it has been found that persons who have suffered injury as a result of exposure to mustard gas may in some cases become hypersensitive to its effects. The condition may be induced by either the liquid or the vapour of mustard gas. It is not possible to say with certainty whether a similar condition may be induced by other types of blister gas, such as lewisite; there is at present no evidence to suggest that this is so.

28. Summary of Dangers of Mustard Gas

The peculiar dangers from mustard gas described in the two previous Sections may be summarised as follows:—

High boiling point and low vapour pressure.	Endow mustard gas with great persistency on all materials.
High freezing point ...	Renders mustard gas comparatively ineffective in cold weather because little vapour is evolved and the liquid is frozen. Under these conditions, however, it is dangerous because it is not detected by the sense of smell; the skin may be contaminated by contact and burned, while the frozen liquid may be carried on boots or clothing into warmer surroundings where it will melt and vaporise.
Stability, i.e., not readily destroyed except by strong chemicals such as chlorine or strong nitric acid.	The chemical stability of mustard gas tends to increase the persistence due to its low vapour pressure, since the substance is not greatly affected by moisture or by contact with most ordinary materials under normal conditions. The result is that articles of the most varied natures may remain dangerous to handle for long periods after contamination, particularly if evaporation of the mustard gas has been in any way partially or wholly inhibited.

Solubility. — Readily dissolves in animal fats, but almost insoluble in water.	Enables mustard gas to penetrate the skin very easily. Not readily destroyed by rain, and difficult to remove by washing with water alone.
Penetration	Like other oily substances, mustard gas readily soaks into most materials other than metals, glass, etc.
Great toxicity ...	Even low concentrations of vapour evolved from the liquid are dangerous to the eyes, lungs and skin. The destructive effects of mustard gas on the tissues of the body are not obvious for some hours after contact. Hypersensitivity may be induced after repeated burns.

29. Nature of Casualties from Mustard Gas Vapour

Mustard gas vapour can be harmful in concentrations that may not be readily noticed by the sense of smell; also, the sense of smell for mustard gas vapour tends to become dulled quickly, in which case the danger may no longer be appreciated.

The cumulative effect of repeated small doses of the vapour is another insidious danger.

The vapour concentration necessary to produce effective results need not be of a high order. One hour's exposure to a concentration of one part of mustard gas vapour in one million parts of air is sufficient to incapacitate an unprotected man for about two weeks through conjunctivitis.

In the event of a mustard gas attack on a large town some persons who have actually been

exposed to the gas may, owing to local conditions, be unable to get away from their places of business before the inflammation of the eyes has become severe. If for that reason they can no longer see they should be treated like blind persons and should be conducted to the first aid post, the hospital, or their homes, as the case may be. The personnel of the Voluntary Aid Organisations should be prepared to give their assistance in such cases, and it need only be pointed out that if this type of casualty is numerous it is possible to collect a small number of them into a party, each man holding on to his neighbour, when a single guide can lead the whole party.

The possibility of the odour of mustard gas being masked by smokes or the fumes from high explosive, and its lack of immediate sensory irritation in what are yet effective concentrations, are additional dangers.

The degree of severity of mustard gas vapour casualties naturally varies with the concentration and the period of exposure. The least severe case may only show light conjunctivitis, with almost no erythema of the skin and only a slight hoarseness of the voice; the most serious, on the other hand, may present a picture of the most profound illness, usually with widespread skin burns, severe eye effects and damage to the respiratory tract.

A moderately severe case of exposure to the vapour when quite unprotected will present a typical appearance in 24 hours, with eye symptoms predominating; general reddening of the skin occurs, most marked in the genital region where the excoriation of the skin may cause distressing irritation, while, at about the same time, the respiratory system begins to show signs of involvement by a partial loss of voice and by a troublesome cough.

In the last war, the death-rate among well disciplined troops with effective respirators was low, approximately 2 per cent. of the mustard gas casualties, but

the death-rate among those without the protection afforded by a satisfactory respirator was much higher. Fatal cases were almost unknown within the first 24 hours after exposure. Death occurred at any date from the second or third day in the most severe cases to the third or fourth week in the more lingering ones, the highest death-rate being at the end of the third or fourth day after exposure; and almost all the deaths were due to secondary broncho-pneumonia.

The main features of mustard gas vapour casualties may be briefly summarised as follows:—

(*a*) An insidious onset, with a latent period of two to 48 hours according to the concentration of the gas and the duration of exposure.

(*b*) Injury to the eyes, varying from simple conjunctivitis of a temporary nature to a severe keratitis and secondary septic complications of a grave character.

(*c*) Laryngitis, involvement of trachea and bronchi, and possibly necrosis of the mucous membrane, leading to severe bronchitis or broncho-pneumonia.

(*d*) Early nausea, or persistent vomiting, accompanied by epigastric pain.

(*e*) Erythema of the skin—early in the case of exposed areas or of hot, moist surfaces—which may proceed to vesication or excoriation, and may be followed by secondary septic infection.

(*f*) Slow healing of the blistered, devitalised areas and pigmentation of the ensuing scar.

The types of injury which might result from exposure to mustard gas vapour are summarised below.

(1) *Action on the Eyes.*

The eyes are usually the first to show signs of the irritant action of mustard gas vapour. Even so the visible onset of injury may be long delayed, the latent period varying from two to 48 hours, according to the dosage; but, once established, it usually develops with

rapidity. The initial symptoms of smarting and irritation are soon followed by lachrymation, pain in the eyes and headache; swelling of the eyelids quickly supervenes and may be so extreme as to close completely the palpebral fissure, while the simple lachrymation becomes muco-purulent as a result of secondary infection, and blepharospasm and photophobia are marked.

Changes in the eyeball itself are equally rapid; the injection which marked the onset of ocular signs is followed by swelling and oedema, to such an extent that the conjunctiva at the interpalpebral aperture may even project between the eyelids, forming a characteristic yellowish-white, opaque band on either side of the cornea. A similar swelling of the palpebral conjunctiva under the eyelids may produce two chemotic folds which add to the distressing appearance of the eye by projecting between the lids.

The cornea, in the early stages, is grey and hazy, the haziness fading off above and below where partial protection is given by the eyelids; its surface becomes blurred and lustreless, and later exhibits a typical " orange skin " appearance. Exfoliation of the corneal cells may occur, and in the presence of trauma ulceration may follow which, if complicated by secondary infection, may lead to permanent opacities and impairment of vision.

In serious cases, the condition of the cornea calls for the most careful and regular examination—a difficult procedure in view of the intense photophobia and blepharospasm. Recovery is slow; the oedema gradually subsides and the corneal epithelium begins to regain its lustre; gradually a condition is produced which is the exact opposite of the original appearance, that is to say, the inter-palpebral area previously a dead white is now once more vascular and goes through a period of injection, whilst the previously injected areas, protected by the eyelids, are regaining their normal tint.

In the absence of corneal ulceration or conjunctival adhesions no permanent after-effects are usually met with, but lachrymation and photophobia are liable to persist for some time, and neurasthenic conditions may supervene in susceptible individuals.

The experience of the last war, when eye casualties were produced by the vapour more often than by a direct splash of the liquid, showed that eye injuries fall into three main groups:—

(*a*) Mild cases, 75 per cent. of the total, fit for duty, on an average, in two weeks.

(*b*) Intermediate cases, 15 per cent., recovery in four to six weeks.

(*c*) Severe cases with corneal changes, about 10 per cent., recovery in two to four months. Of these only a very small minority sustained total loss or impairment of vision.

(2) *Action on the Respiratory Tract.*

The toxic effects of mustard gas vapour on the respiratory tract are shown by an early rhinitis (almost simultaneous with the onset of the conjunctivitis), accompanied by sneezing and the discharge of a profuse watery secretion, soon to become muco-purulent.

The larynx is usually affected early, and hoarseness or aphonia is frequent. The laryngitis may be mild if exposure has been limited to a low concentration, but oedema and even sloughing of the vocal cords may follow exposure to a high concentration.

In a severe case, the laryngeal inflammation tends to be reproduced in the trachea and bronchi, when the dry irritating cough, originally complained of at the onset of the laryngitis, is replaced by a loose cough accompanied by profuse muco-purulent expectoration and pain behind the sternum. A rising temperature and pulse indicate the onset of a severe bronchitis which may be complicated by sloughing of the inflamed

tracheal mucous membrane; secondary infection of the latter soon leads to the development of a broncho-pneumonia with cyanosis. Rarely, abscess of the lung, bronchiectasis, or even gangrene of the lung may occur—not as a direct result of the gassing by mustard gas vapour, but of the secondary bacterial invasion which follows.

In the great majority of cases, however, the lesion is confined to a bronchitis which clears up in the course of a month or six weeks, leaving no after-effects.

(3) *Action on the Skin.*

Before describing the effects of mustard gas vapour on the skin it may be useful to mention some of the factors that influence the penetration of the gas or modify the severity of its action.

As in the case of liquid mustard gas, the vapour owes its penetrative powers to its ready solubility in the lipoid constituents of the skin. The degree of skin burning which follows is accentuated if the exposed skin area be a highly sensitive and tender region such as the scrotum, or if it be a surface which is subjected to constant friction, as is the case in the neck, the wrist and the ankles.

If the exposed skin surface be bare, the attack of the vapour will be direct, and the result more rapid than if the skin be clothed. This temporary protection of clothed areas is due to the fact that ordinary porous clothing material absorbs the vapour and retards its access to the skin; but if such clothing be worn beyond the period of actual exposure, or if the exposure be prolonged, the vapour retained by the clothing will increase the severity of the resulting skin burns.

This temporary protection varies in duration according to the nature, texture, thickness and degree of humidity of the clothing. Thus, a thin openwork cotton garment in close apposition with the body surface will

not greatly retard the access of the vapour to the skin, whereas thick close-woven material, such as serge and woollen clothing generally, will definitely do so, and may even save the area from burns provided it be discarded on leaving the contaminated area.

After the lapse of the usual latent period, which may vary from 2 to 48 hours after exposure to the vapour of mustard gas, an erythematous blush appears over the affected area and gradually deepens in intensity until the skin looks scorched. This redness is not unlike the eruption of scarlet fever, and is usually accompanied by only a slight degree of irritation. The erythema is most marked on the skin areas which are hot and moist; dense tissues like the scalp, the palm of the hand or the skin of the heel usually escape unless the concentration of the vapour be high and localised to that area, as, for example, from drops of liquid mustard gas on a cloth cap.

The affected area soon begins to show superficial blistering in the form of small vesicles which rapidly coalesce to produce large blisters full of a clear, yellow serum: on evacuating this fluid and removing the overlying epithelium, a raw, red, weeping surface is exposed. As a rule vesication is complete by the second day, but blisters may appear in crops for days following exposure, even though all contaminated clothing was discarded at an early stage. Systemic disturbance is absent, unless the burns are extensive and severe; interference with sleep, however, may be caused by the distressing itching which may accompany the developing burns. Very mild cases may show simply erythema, followed later by pigmentation with scurfy desquamation, the " blister " stage being absent.

In severe cases the erythema may deepen to a dusky, almost violet tint, oedema of the skin is marked, and blisters appear in the dark background overlying a deep red or haemorrhagic base. Such blisters progress slowly, and are very prone to sepsis owing to the

serious devitalisation of the tissues; ulceration is liable to spread beyond the limits of the blister, and healing is very slow. If sepsis occurs it adds to the severity and duration of all lesions; the necrosed tissues form an excellent medium for pathogenic organisms, and death may result if extensive or deep burns are thus affected.

The healing of an uncomplicated vapour burn is more rapid than one due to liquid mustard gas, but a common feature of all mustard gas burns is the long time they take to heal. The chemical irritant seriously damages the vitality of the affected tissues, and all processes of skin repair are delayed.

The healing stage is characterised by a brownish or coppery pigmentation of the epithelial layers in the areas previously affected by the erythema. This staining, however, is superficial, and usually disappears with the normal desquamation of the superficial layers of the skin.

As a rule, serious after-effects are absent, and the scars resulting from vapour burns are shallow, but a chronic eczematous condition or a generalised furunculosis may, rarely, follow such burns and prove obstinate to treatment.

(4) *Other Effects of Mustard Gas Vapour.*

Apart from its direct action on the eyes, the respiratory organs and the skin, mustard gas vapour may indirectly, and mainly owing to secondary infection produce signs and symptoms in more remote organs of the body:—

(a) *Alimentary tract.*—It has already been mentioned that an early nausea, or even vomiting, accompanied by epigastric pain, often occurs in vapour poisoning by mustard gas. This effect is due to the swallowing of saliva or nasal secretion impregnated with the gas. Although it may prove obstinate during the first day, it rarely persists

for more than 48 hours; similarly, the accompanying epigastric pain is of short duration, and the intestines are not usually affected. There are no lasting after-affects, but a functional condition of persistent nausea or vomiting has been observed occasionally.

(b) *Urinary tract.*—Traces of albumen have been found in the urine of early fatal cases, most probably due to congestion from circulatory weakness and not as a result of the action of the gas on the kidneys. It is only in the late stages of fatal cases, and particularly in those instances where widespread septic burns have occurred, that renal complications have been noticed, such as an acute haemorrhagic nephritis. Pain on micturition, however, and even retention of urine may result from a local oedema and vesication of the penis.

(c) *Circulatory system.*—Blood changes are not met with, and any alteration in the leucocyte count is due either to pneumonic complications or to sepsis of the skin burns. Apart from the effects of the general toxaemias of pulmonary sepsis, no primary changes occur in the cardiac mechanism, but a functional condition of disordered action of the heart is not uncommon as a result of the gassing.

30. Nature of Casualties from Liquid Mustard Gas

The great majority of mustard gas casualties in the last war were caused by exposure to the vapour emanating from collections of the liquid deposited by shell. Aircraft bombs, owing to their different methods of construction, can contain more liquid gas than shells of the same total weight, and in addition aircraft can discharge liquid mustard gas as spray. Casualties in future wars may therefore be of more serious types caused by contamination by the liquid itself.

Casualties from the liquid may result in the case of:—

(i) persons in the open under falling spray;

(ii) persons near enough to the burst of a bomb to receive direct splashes of the liquid; and

(iii) persons who touch material objects which have splashes of the liquid upon them.

The types of injury which might result are summarised below.

(1) *Skin Burns due to the Liquid.*

(a) *On bare skin.*—Although liquid mustard gas is a direct irritant to the skin, the sensory irritation is not immediate; its high lipoid solubility enables it to penetrate tissues rapidly, but hours may elapse before the clinical signs make their appearance.

Penetration is rapid, and this rapidity is enhanced by an elevated temperature of the skin, or under hot weather conditions. Indeed, there is reason to suspect that constant exposure to heat, as in tropical or semi-tropical countries, leads to the acquisition of some degree of sensitivity to mustard gas.

The initial signs and symptoms of a typical mustard gas burn are an erythema at the site of contact, often accompanied by some itching; the capillaries become engorged, and oedema, with thickening of the skin, supervenes. The erythema deepens, and in severe cases may even assume a livid hue; a pale, parchment-like area makes its appearance in the centre of this erythematous zone, and a vesicle, tensely filled with clear, yellow serum, gradually forms. This vesicle is the result of an inflammatory exudation of fluid which may continue for several days, according to the depth of penetration of the liquid mustard gas; the exudate, however, contains no actual mustard gas.

If the liquid contamination of the skin be widespread, as in a smear or splash, the erythema is followed by the appearance of numerous small vesicles

which gradually coalesce to form large blebs, the underlying area being raw and oedematous; such blisters may continue to develop in crops for several days after contamination.

There is no evidence that any of the liquid mustard gas finds its way into the general circulation. Apart from the itching—which may be most severe where warm moist parts of the body are affected—there is little or no irritation except some stinging while vesication is developing, and no pain follows the appearance of the latter. The danger of sepsis following, however, is a real one, especially if the blistered area be extensive, as the tissues affected are devitalised, and the blood supply is impaired.

In the absence of secondary infection no constitutional disturbance is usually noted, and primary shock is absent. Healing, however, is a slow process (partly because the blood supply has been damaged, and partly on account of residual mustard gas or its derivatives persisting in the tissues). The resulting scar, which is soft and pliable, often assumes a coppery pigmentation which disappears after a time.

(b) *On clothed skin.*—Drops of liquid mustard gas on clothed areas of the body act by virtue of the high concentration of vapour evolved, the warmth of the underlying skin naturally assisting the process. A gross contamination of the clothing, on the other hand, such as may be produced by splashes or by accidental spilling, may result in actual contact of the liquid with the skin, when the action of the vapour would be superadded to that of the liquid.

All ordinary clothing is pervious to liquid mustard gas; but it is obvious that penetration will be much more rapid in the case of the single thin cotton garment of tropical and sub-tropical countries than with the multiple layers of woollen clothing worn in temperate climates.

If the garments be damp or wet, small drops of liquid mustard gas will readily penetrate and burn the skin. Although no reasons can be given for this, it has been proved experimentally to be an accurate statement of fact.

(2) *Eye Burns due to the Liquid.*

Contamination of the eye by spray or splash represents one of the gravest dangers to which the body can be subjected in the presence of liquid mustard gas, as permanent damage will result.

The degree of discomfort which immediately follows contact of the liquid with the eye may be slight, and usually subsides; symptoms often commence within half-an-hour, however, and within an hour or two the eye is inflamed and the eyelids are swollen and painful.

The clinical signs are ushered in by profuse lachrymation and conjunctivitis, and the condition develops with great rapidity. The eyelids become painful, swollen and greatly thickened by oedema, the palpebral conjunctiva is red and oedematous and the cornea develops opacities, while the ocular conjunctiva becomes congested and shows signs of ulceration. Intra-ocular tension is increased, pain and headache are severe and a muco-purulent secretion exudes from the closed eyelids. Photophobia and blepharospasm may be extreme, and great difficulty is encountered in examining the swollen and painful eye.

Following actual liquid mustard gas contamination of the eye, large areas of the conjunctiva may readily be shed, and partial or complete loss of vision results from the extensive ulceration and subsequent scarring.

Persons who have suffered from severe liquid contamination of the eye are liable to a recurrence of the symptoms on the slightest abrasion even up to 20 years later. This is probably due to the devitalised condition of the eye.

31. Need for Preventive Treatment in case of Mustard Gas Contamination

In reading what follows on the treatment of persons contaminated by mustard gas, it should be remembered that many of these may also be suffering from wounds or physical injury. Such casualties will have to be dealt with according to the particular circumstances of the case, but the treatment for contamination should follow that laid down in the later sections of this chapter so far as is compatible with the nature of the wounds. Clothing should be completely removed, and the patient himself thoroughly cleansed in order to remove the contaminant, before the wounds are dressed.

Preventive treatment consists essentially in the speedy and complete removal of all contaminated clothing and in freeing the skin from the contaminant, whether liquid or vapour.

Exceptions to, or modifications of, this general rule may be met with, as, for example, in the case of a small localised liquid contamination of the bare hand, or after exposure to a low concentration of the vapour, when prompt local cleansing of the skin or a change of clothing respectively will suffice.

In view of the rapid penetration of the skin by mustard gas, treatment should not wait until a doctor is called, and it is part of the training advocated in A.R.P. Handbook No. 1 (Personal Protection against Gas) that all members of air raid precautions services, and as far as possible the general public, should be taught to undertake treatment for themselves. Nevertheless it is a matter of importance that it should be thoroughly understood by doctors.

Each case will have to be considered on its merits; but, whatever the type or extent of the contamination, *speed is the essence of all preventive treatment.*

Delay of a minute or two in the case of liquid contamination, or of ten to fifteen minutes following exposure to the vapour, before cleansing of the skin is undertaken enhances the danger and may result in definite burns of the affected areas.

When the skin is hot as a result of exercise, and in hot or tropical countries, the results obtained by all preventive methods of decontamination of the skin are inferior to those obtained when the skin is cool and dry, and the need for prompt action is even greater.

After removal of all contaminated garments (which must not be used again until decontaminated) preventive treatment of the skin should be undertaken without delay. The choice of methods is not large, but one or more of them should be readily available at all times. The method adopted must be that which can be most promptly applied.

32. Preventive Treatment for Contamination from Mustard Gas Vapour

After contamination with the vapour of mustard gas —i.e., after exposure to an atmosphere contaminated with the gas, or when the outer clothing has been sprayed, or has otherwise come in contact with the liquid form of the gas—preventive treatment should consist of a rapid removal of all clothing, followed as soon as possible by a thorough washing of the whole body surface with soap and water, preferably under a shower.

Lavage of both eyes with warm water or normal saline should be carried out as soon as possible, and should be repeated every two hours. Similarly, the effects of vapour contamination of the nasopharynx may be minimised by prompt irrigations.

Attention is directed to the paragraph at the end of Section 10, describing the fundamental differences between affections of the eyes by lachrymatory and by vesicant gases.

33. Preventive Treatment for Liquid Mustard Gas Contamination

The following methods are possible:—

(a) *Bleach treatment.*—Thoroughly rub into the affected area, for a minute or so, either bleach ointment or other approved protective ointment, or aqueous bleach paste. This procedure chemically neutralises the mustard gas.

As a first step in the prevention of burns, when the contamination is small and localised, thorough rubbing with the ointment is the method of choice. For extensive contamination by the liquid, however, a thorough inunction with aqueous bleach paste will be found more easy of application.

When the operation is completed the ointment should be wiped off, or, if the aqueous paste was used, the affected part should be flushed with water—the object being, in each case, to remove surplus bleach from a potentially injured area. Bleach will destroy free mustard gas quickly, but it will also irritate the skin if left in contact with it. Care must be taken to prevent access of bleach to the eyes.

Bleach should *not* be used if an erythema has already developed, as it aggravates the condition.

Actual vesication of the skin by drops from mustard gas spray may be avoided if preventive treatment be undertaken within a minute or two after contamination. Even though the delay be longer, bleach will still be the method of choice so long as liquid mustard gas is visible on the skin, as it will mitigate the severity of the resulting burn.

Bleach ointment is made by mixing equal parts, by weight, of "supertropical" bleach and white mineral jelly, while the aqueous bleach paste consists of "supertropical" bleach mixed to a creamy consistency with water—roughly, one part of bleach to

one or two parts of water by volume. The ointment keeps well in temperate climates, while the aqueous paste retains its effectiveness for several days if it be stored in enamelled containers with well fitting lids; for tropical climates a special protective ointment is desirable.

Bleaching powder is ordinary chloride of lime, while " supertropical " bleach is the same substance stabilised by the addition of quicklime, and fulfilling certain conditions of stability and chlorine-content.

Ordinary bleach is more irritating to the skin than the supertropical variety, but in the absence of the latter is suitable for preventive treatment when made up as an ointment with white mineral jelly, or into a paste with water, provided prolonged storage is not required.

The use of *white* mineral jelly is essential: yellow mineral jelly in contact with bleach may generate heat, and may even produce combustion on storage. If mixing is carried out in bulk, the employment of a mill is advocated in order to ensure a thorough and uniform consistency.

(b) *Removal of contamination by means of a solvent.*—Swab the contaminated area repeatedly with *petrol, kerosene, carbon tetrachloride* (but see Section 56), *or other solvent of liquid mustard gas.* It is important to remember that these solvents do not destroy the gas, but merely dissolve it; hence the swabbing must be confined strictly to the contaminated area, and must be repeated.

This method is effective if carried out by skilled individuals, and solvents are within easy reach; certain precautions, however, are very necessary. Oilskin or rubber gloves must be used if available; otherwise, the swab should be only partly immersed in the solvent, and it should be held between finger and thumb by the dry portion, or preferably in forceps; the wet portion is then applied to the contaminated

skin so as to soak up the liquid contamination, care being taken that none of the solvent runs over the skin of either the subject or the operator; the contaminated swab is then discarded and the process is repeated for several minutes with fresh swabs, or as long as the characteristic odour of the gas persists on the skin. Thorough washing with soap and water, if available, will complete the treatment. The contaminated swabs, must, of course, be destroyed by burning and the gloves and forceps decontaminated.

One disadvantage of this method in the hands of unskilled persons is that the solvent is apt to " run " on the skin and cause burns on areas comparatively far removed from the original site of contamination; a further disadvantage is the liability of the operator's fingers to become contaminated in the absence of gloves. Employed with care and intelligence, however, the method is valuable in an emergency.

(c) *Thorough washing.*—Wash thoroughly the affected part with *soap and water,* using frequent changes of water. This process does not destroy the mustard gas, but merely removes it in the lather; the scrubbing must, therefore, be confined to the contaminated area, and the hands should be safeguarded, if possible, by suitable gloves.

If the liquid contamination be small, localised and of known situation, this is an effective method of removing it if carried out promptly. In any case, vesication of the skin is usually prevented if the treatment is not delayed beyond five minutes, though an erythema will probably result.

With a gross contamination, or when the drops of liquid mustard gas are multiple, the results of scrubbing with soap and water are unfavourable, as it is difficult to avoid spreading the contaminant in the soapy lather to surrounding areas. Under these circumstances bleach treatment is the method to adopt if available.

Should it not be possible, however, to deal with such a contamination until some time has elapsed, thorough washing should still be carried out at the first available opportunity in the hope of mitigating the degree of burning.

(d) *Special treatment for eyes.*—Apart from the skin surfaces, the only other areas to which preventive treatment can be extended are the eyes.

Contamination of the eye by liquid mustard gas presents a very serious problem. Should an eye be contaminated by the liquid, however small the drop may be, immediate preventive treatment should be undertaken. None of the methods recommended for the skin is applicable for this purpose; simple, but rapid, removal of the contaminant by bland, unirritating methods is indicated.

This may be done by thoroughly flushing out the conjunctival sac with warm, plain water, or some bland solution, after opening the eyelids wide. This flushing should be most thorough and should be repeated hourly in the hope of mitigating the damage to the eye. If evidence of local irritation appears, a drop of liquid paraffin or castor oil should be instilled to prevent the eyelids adhering. Cocaine is contra-indicated.

34. Curative Treatment for Mustard Gas Casualties

The first essential in the treatment of mustard gas casualties is the prevention of further infection from contaminated clothing; it will be necessary, therefore, to strip the patient completely and to wash the entire body surface, employing frequent changes of soap and water. The opportunity should also be taken at this stage to douche the eyes thoroughly.

The second essential is to relieve immediate symptoms.

The question of treatment is best dealt with by taking in succession the various parts affected.

(a) *Treatment of the Eyes.*

Although *liquid* contamination of the eye may produce some irritation on contact, this usually subsides and may be followed by an absence of symptoms lasting about half-an-hour. Within one hour, however, the eye is red and swollen, and the lids are half closed. It should be unnecessary to stress the futility of waiting for such signs, or for subjective symptoms to appear, before undertaking systematic treatment. The risks attending liquid contamination of the eye are so grave that any history of such an accident should be sufficient to justify immediate and thorough treatment.

At this early stage treatment is limited to thorough and frequent lavage of the eye with a warm 2 per cent. solution of boric acid, or normal saline solution, in the hope of mitigating the severity of the inevitable lesion.

After the onset of clinical signs, treatment is largely symptomatic, and in the earlier stages will be confined mainly to the relief of pain and to free irrigation; the latter, however, will present some difficulty owing to the intense photophobia and blepharospasm which exist, and the general oedema which pervades the tissues.

When spasm and pain are marked the application of sterilised 1 per cent. atropine ointment (or perhaps an aqueous solution or lamellae) every 12 hours will give relief, and in all cases where the cornea is affected this treatment should be persisted in. Cocaine should not be used to allay the pain, as this drug, which exerts only a transient anaesthetic action, tends to loosen the corneal epithelium and facilitate ulceration.

Free drainage of the discharge is essential, and on no account should the eye be bandaged as this will only result in damming back the secretions with

disastrous results. Shades of brown paper or other light material may readily be improvised to relieve the photophobia, and a few drops of sterilised liquid paraffin may be inserted several times a day to prevent the eyelids becoming glued together and impeding free drainage.

When the discharge becomes muco-purulent the instillation of a weak (2 per cent.) solution of argyrol or protargol twice daily will be found useful. This treatment is of particular importance when the cornea is grey and roughened, in order to avoid the danger of an infiltrating ulceration. Should this occur, the ulcer may be cautiously cauterised by the light application of pure carbolic acid put on with a nearly dry brush slightly moistened with the liquid. Frequent bathing and hot applications over the closed lids four times a day will assist in relieving pain.

If hypopyon supervenes and does not clear up with hot bathing, atropine and frequent cleansing of the conjunctival sac, Saemisch section is indicated.

With *vapour* contamination of the eye the prognosis is very much more favourable, and it is important that the patient be reassured from the outset that his eyesight will not be lost. Treatment, however, must be prompt and assiduous, as all contamination of the eye, however light, is a prolific source of invalidism.

For mild cases, where exposure to the vapour has been of a short duration, frequent lavage or warm irrigations every two hours will suffice to clear up the condition. The instillation of a few drops of liquid paraffin will prevent the tendency of the eyelids to adhere, and a quiet, darkened room or an eyeshade will materially add to the patient's comfort if any degree of photophobia be present. An astringent lotion and general tonic treatment will complete the cure.

In more severe cases, however, both pain and spasm may be marked, and the cornea may be affected. Under these conditions the treatment should be on the lines of that recommended for cases of liquid contamination, the primary indication being the prevention of corneal ulceration or the formation of adhesions.

(b) *Treatment of the Respiratory Tract.*

The early *rhinitis* is usually overshadowed by the condition of the eyes; should there be pain and distressing discharge it may be treated with copious warm douches of sodium bicarbonate in 5 per cent. solution several times daily. In the rare cases where a persistent muco-purulent discharge, associated with ulceration and occasionally with epistaxis, is long continued, an astringent lotion containing zinc sulphate with boric acid will be found helpful.

Laryngitis.—The laryngeal irritation is best dealt with by topical treatment such as laryngeal spraying or by the inhalation of steam from a pint of boiling water containing a teaspoonful of a mixture of menthol grs. 10 in 1 oz. tinct. benzoin. co.

Broncho-pneumonia.—As the majority of deaths from mustard gas in the last war were due to secondary infections of the respiratory tract, treatment should be directed, from the outset, towards combating bacterial invasion of the bronchi.

As a preliminary step against extraneous infection, all cases of mustard gas poisoning in which the respiratory tract is involved must be kept apart from other patients suffering from infective pulmonary disorders; they should, if possible, be segregated in special wards, and the onset of broncho-pneumonia in one of them should entail his isolation.

The routine employment of volatile antiseptics from the earliest stage will be facilitated by the adoption of a pliable, perforated mask, fashioned in the form

of a Burney Yeo inhaler, containing a pad of gauze on which a few drops of the antiseptic are placed hourly. A useful formula is the following:—

Menthol	gr. 20
Chloroform	min. 60
Creosote	min. 60
Ol. Eucalypti	min. 20
Tinct. Iodi	min. 30
Sp. Vini Rect.	to one ounce

The value of menthol in mustard gassing is enhanced in those cases which require operative treatment for some concomitant wound. In these cases the laryngitis is such that, until it has been allayed by the inhalation of menthol, it may be impossible to induce anaesthesia, as the anaesthetic sets up paroxysms of coughing.

In the various stages of the broncho-pneumonia, treatment is symptomatic and follows the recognised rules of procedure, including the employment of expectorants where the muco-pus is tenacious and difficult of expulsion. It may be stated here that the prophylactic venesection advocated for phosgene cases, which is of value in the early treatment of pulmonary oedema, has no place in the treatment of mustard gas cases, though occasionally it may be indicated at a later stage to relieve the right heart of embarrassment and cyanosis induced by a diffuse broncho-pneumonia. The same may be said of oxygen therapy, which, although essential in the pulmonary oedema caused by phosgene, is only indicated occasionally and at a late stage in mustard gas poisoning when a condition of oxygen want is established as the result of grave and widespread pulmonary damage.

(c) *Treatment of the Skin.*

As in other regions of the body, septic infection is the most potent factor in delaying the satisfactory healing of skin burns. When it is remembered that

mustard gas penetrates, and in so doing devitalises, the skin, it is obvious that early preventive treatment is of paramount importance, inasmuch as it will lessen the severity of the skin burns and reduce the risk of sepsis, and that any curative treatment should have some antiseptic value.

As a preliminary to all local treatment it is essential to cleanse the skin as thoroughly as its damaged condition permits, and to clip short all hair, if any, on the affected area. *It may be useful to repeat here that the application of bleach in any form to a skin which is already showing signs of damage will aggravate the ensuing burn.* It must also be noted that skin surfaces damaged by mustard gas are exceedingly susceptible to trauma, and that even the continued pressure of an ill-fitting bandage may lead to an extension of the damage.

As treatment will vary according to the nature and degree of the burns, it will be best to consider these in detail:—

(i) *Erythema.*—Mild cases which do not proceed beyond an erythema heal spontaneously, with possibly some desquamation and pigmentation. They may be compared to sunburns in severity and discomfort, and clear up just as readily. If the skin is unbroken a mildly antiseptic dusting powder may be applied.

(ii) *Vesication.*—It is this stage that will afford a critical test of successful treatment through the elimination of secondary infection, as the devitalisation of the tissues in these cases is much more profound.

Any available cleansing treatment in use in surgical practice will suffice for the undamaged skin surrounding the burn itself. In the last war extensive use was made of Eusol and of Dakin's solution for the treatment of burns, but they are too painful for continued use on raw surfaces. Picric acid and similar powerful

germicides are undesirable because of the toxic symptoms that may follow their absorption, while ointments and pastes are, as a rule, contraindicated because of their tendency to seal up discharges; for the same reason powders are undesirable as they are apt to produce crusts which retain the discharge.

When discrete, circumscribed blisters make their appearance they should be evacuated, under aseptic conditions, by means of a hypodermic syringe or a sterile needle, gentle pressure being applied, if necessary, upon the walls of the blister with a sterile swab to ensure complete evacuation; the intact epithelium should then be allowed to collapse and seal down the raw, sensitive surface underneath. This evacuation of fluid from blisters may have to be repeated owing to the continued oozing of serum from the raw area; if this procedure be delayed some hours the serum may be found to have coagulated, in which case the overlying epithelium should also be removed. The further treatment of these circumscribed vesicles consists in the application of dry dressings.

Satisfactory results have followed the use of crude cod liver oil in the treatment of comparatively small mustard gas burns after evacuation of the blister and removal of dead skin: the healing of the burns was rapid. The oil is freely applied on lint, which is then covered by a pad of cotton wool. The dressings are changed daily; little or no irritation is caused, and the oily dressings come off easily and without pain.

When larger areas are affected, however, and when the blisters are confluent, better results will follow the use of a non-irritating antiseptic such as " Dettol ", made by adding 20 per cent. by volume of " Dettol " to a freshly prepared 5 per cent. solution of tannic acid. After evacuating all blisters and removing the loose epithelium, the solution is applied directly to the raw surfaces either as a spray or, preferably, on lint, as a coagulum appears to form more quickly

on a moist dressing than when an atomiser is used. Three or four layers of the lint are soaked in the mixture and applied to the burnt area, which is then covered lightly with cotton-wool and a gauze bandage; the cotton-wool and bandage may be removed every two or three hours, but the lint is left in position and is re-soaked. The entire dressing may be removed at the end of 8 to 12 hours, by which time a firm coagulum has formed; this is then sprayed with 5 per cent. tannic acid solution and dried.

A further step towards the reduction of possible infection may be taken by swabbing a large area surrounding the burn with the antiseptic, for the sepsis which sometimes occurs at the edges of the coagulum appears to originate from the surrounding skin; further, it is advisable to repeat this swabbing every four or six hours until the coagulum has separated.

After the separation of the coagulum, the general principles of wound treatment are applied to the unhealed areas which remain; stimulating lotions or scarlet red ointment will be found of use in encouraging the growth of new epithelium.

In cases where the condition is already septic, continuous baths, at body temperature, of a mildly antiseptic nature will prove both soothing and efficacious, while hot hip baths of isotonic salt solution are helpful in allaying the intense irritation of mustard gas burns of the groin and genitalia. If hot compresses or fomentations be employed, lint should be used in preference to gauze as it is less painful to remove; oiled silks should be avoided, as they keep the burns sodden and retain the discharge.

35. General Treatment for Mustard Gas Casualties

Where nausea, vomiting or epigastric discomfort is present, the diet should be light and fluids may be given freely; should these not be retained, the administration of 10 to 20 grs. of sodium bicarbonate may be of assistance, and the patient should be encouraged to

drink water freely. As convalescence proceeds, and in all cases of uncomplicated body burns, a full diet is required, and this should be as varied as possible. Cases showing evidence of commencing fever, which may be a prelude to broncho-pneumonia, should be suitably dieted.

Experience has shown the importance of combating functional after-effects. Functional disorders fall, in the main, into two classes. In the first, exposure to gas, often to a minimal and barely toxic concentration, may yet prove the final factor in upsetting a nervous system already breaking down as the result of physical or mental strain. In such circumstances, and especially when combined with ignorance, it may produce an "anxiety state" similar in all respects to the neurosis so common in the last war.

The second class is a more important one, because in these cases a local, but real, organic lesion from mustard gas causes certain irritant reflexes, such as coughing or photophobia, and these sensory reflexes are perpetuated by introspection, almost in a form of conversion hysteria, long after their organic cause has been cured. Lack of appreciation of this possibility by doctors will cause much delay in discharging casualties.

Functional photophobia and aphonia are responsible for the great majority of cases. This is not surprising when it is realised that the initial trauma affects a highly organised special sense, and that fear of blindness or dumbness resulting from the injury may very well act to prolong the symptoms. Ill-advised and unnecessary treatment, however, is also a probable factor in many cases, as, for example, the continued retention of eye-shades long after the necessity for them has passed and the actual lesions have totally disappeared. There can be no doubt that the suggestive influence of wearing a shade under these conditions will prolong the functional manifestation.

Persistent aphonia, often accompanied by a useless, harsh cough, is another striking evidence of auto-suggestion arising from the initial laryngeal irritation. The characteristic cough is either dry, or accompanied by watery sputum mainly of salivary origin; it is usually much worse at night, and is of a ringing, harsh quality. If the doctor realises the nature of the condition and gives the patient confidence in his early recovery, this functional aphonia yields very rapidly to treatment by suggestion and breathing exercises.

Of all after-effects, functional or organic, those which seem to affect the heart present the greatest difficulty in assessment. Disordered action of the heart ("D.A.H." or effort syndrome), with its shortness of breath and tachycardia following exercise, arises from so many diverse causes that gassing was naturally regarded as one of them. It is clear, however, that under competent medical treatment, the incidence of "D.A.H." in mustard gas casualties should be very low provided that serious complications, such as broncho-pneumonia, have been absent.

36. Invalidism after Mustard Gas Poisoning

Experience from the last war showed that the chemical damage to the skin, to the respiratory passages, and to the outside of the eyes might cause prolonged devitalisation of these tissues and a poor resistance to secondary bacterial infection, but no deeper trouble resulted. There has been no subsequent evidence of the irritation leading to later malignant changes in any tissue. The skin ultimately regained perfect vitality. The trachea and bronchi in some cases showed a tendency to relapses of bronchitis, but there was no special proneness to pulmonary tuberculosis. In a few, and fortunately very rare, cases the cornea never regained its natural

vitality, and after even 15 or 20 years a superficial abrasion and infection might spread rapidly and cause permanent loss of sight.

Invalidism in general was not prolonged, but it should be remembered that the casualties dealt with in the war were very largely from exposure to mustard gas vapour and only rarely had they been affected by direct splashes of the liquid. In all cases admitted to hospitals there was some degree of conjunctivitis and laryngitis as well as skin burns. The skin lesions from vapour healed quickly, usually in less than a month. Prolongation of invalidism was due rather to trouble in the respiratory passages and eyes, and to general debility. By following up the times of recovery in a large number of cases at convalescent depots in France, it was proved that at least 75 per cent. of mustard gas casualties admitted to hospitals on the lines of communication, these being the severer cases evacuated from the army zone, could be returned to full duty in less than eight weeks. This involved an average stay in hospital of two to three weeks, during the last half of which time the casualties did not require to be in ward beds or to be specially attended by nursing orderlies.

The worst cases might remain in hospital for two months or even longer. Photophobia, either functional or associated with a tendency to recurrent keratitis, often lingered. Next as causes of invalidism came bronchitis and laryngitis, and lastly D.A.H. and neurasthenia or some general debility. But out of a group of nearly 800 severe cases detained in hospital beyond the ninth week, none died and ultimately only 0·5 per cent. were discharged as permanently unfit for service. The ultimate invalidism from mustard gas vapour was therefore very small. As in the instance of phosgene poisoning, it is probable that any persistent chest trouble was due to mischief wrought by the smouldering inflammation of secondary

bacterial infections rather than by a direct chemical action. Doctors should realise that gas poisoning, whatever the chemical irritant concerned, does not in itself cause a permanent poisoning of the patient or chronic impairment of his health. It is necessary to insist on this truth lest the patient be allowed to develop a morbid dread, and drift into neurasthenia and general debility.

CHAPTER VII

LEWISITE

37. General Description of Lewisite

Lewisite, or chloro-vinyl-di-chlor-arsine, is an important chemical compound developed towards the end of the last war, so that knowledge of its action rests on laboratory experience rather than on the study of actual war casualties.

In the pure state lewisite is a powerfully toxic substance, embodying the aggressive qualities of the asphyxiant gases, the irritant characteristics of the tear and nasal irritant gases, and the universal action on all contaminated tissues of the blister gases. Under modern conditions of chemical warfare, however, its vesicant action would predominate.

Broadly speaking, the effects of lewisite on the human body are similar to those produced by mustard gas, but as it has an immediate irritant action both on the respiratory tract and on the skin, it is more easily detected. In order to avoid repetition, therefore, the following remarks will be confined mainly to salient points of difference in the pathological effects of the two vesicants.

It is assumed that, as in the case of mustard gas, lewisite could be used either in bombs or as spray.

38. Physical and Chemical Properties of Lewisite

A study of the main physical and chemical characteristics of lewisite will be found of assistance when comparing its action on the body with that of mustard gas.

Appearance.—Lewisite is a heavy, oily liquid, colourless in the pure state, but darkening on standing. The impure form, which is more likely to be used in war, is dark in colour.

Odour.—Pure lewisite has no smell, but is extremely pungent and irritating to the nose, giving rise to symptoms of coryza, with sneezing and lachrymation. On contact with moisture or in the impure form, it possesses an odour very closely resembling that of geraniums. This characteristic smell, coupled with the extreme irritancy of the vapour, renders lewisite easy of recognition even in the presence of other gases.

Boiling and Freezing Points.—The boiling point of lewisite is high, viz. 190° C. (374° F.); hence, like mustard gas, it is classed among the persistent chemical warfare agents. Unlike mustard gas, however, lewisite has a low freezing point (about −13° C. or 8·6° F., as opposed to 14·4° C. or 57·9° F. respectively for pure mustard gas)—a difference of practical importance, inasmuch as lewisite will still be an effective liquid in very cold weather when mustard gas may be frozen solid and its aggressive action in abeyance.

Solubility.—Lewisite is insoluble in water; contact with water results in hydrolysis of the compound with the production of hydrochloric acid and organic oxides—a process which is greatly hastened by an elevated temperature. Lewisite is readily decomposed by alkalies. It is, however, freely soluble in the ordinary organic solvents, in the petroleum series of hydrocarbons, and in oils and fats. As in the case of mustard gas, the lipoid solubility of lewisite endows it with its characteristic property of rapid penetration of the skin.

Stability.—Lewisite is much less stable than mustard gas from a chemical point of view, although, in the absence of hydrolysis, it retains its vesicant properties for a considerable time. Hydrolysis in the presence of hot water or an alkali is rapid. As with mustard gas, strong oxidising agents or chlorine will neutralise lewisite, though not so readily.

Penetration.—Like mustard gas, lewisite possesses powerful penetrative properties which, added to the persistent quality of the gas, enable it to render clothing and other materials contaminated by it dangerous to wear or to handle.

39. Toxicity of Lewisite

Lewisite is both an asphyxiant and a vesicant, more rapid than mustard gas in its action, producing more discomfort on inhalation and more irritation when placed on the skin. It thus lacks the insidious character of mustard gas inasmuch as its extreme pungency and penetrative odour ensure its early detection when similar concentrations of mustard gas might not be noticed.

Like mustard gas, lewisite will attack any part of the body exposed to it. Its vapour is a powerful irritant of the whole of the respiratory tract, and, under suitable conditions, may produce vesication of the skin; in the liquid form it penetrates the tissues rapidly and, after a short latent period, gives rise to severe blistering. Liquid lewisite in the eye entails immediate incapacitation.

In contrast with mustard gas contamination, which is never followed by a general systemic poisoning through absorption of the chemical, a massive skin contamination by liquid lewisite may produce acute arsenical poisoning in which all the organs of the body may be affected and arsenic may be found in all the tissues.

As mentioned in Section 31 for mustard gas cases, the situation of persons contaminated by lewisite may be aggravated by wounds or other physical injury.

There is no evidence, as yet, of the acquisition of a hypersensitivity after repeated burns by lewisite—a condition which has been proved to occur occasionally after similar mustard gas exposures.

40. Comparative Dangers of Lewisite and Mustard Gas

Lewisite and mustard gas may be compared as follows:—

(*a*) The easy detection of lewisite as compared with mustard gas tends to reduce its effectiveness.

(*b*) Although lewisite with its higher vapour pressure tends to produce a higher concentration of the vapour than mustard gas under similar conditions, the effective duration of such a concentration is shorter.

(*c*) The rapid hydrolysis of lewisite in the presence of moisture renders it less suitable than mustard gas for purposes of ground contamination, or for use as a spray in moist climates.

41. Nature of Casualties from Lewisite Vapour

The irritant character of lewisite vapour, even in low concentrations, ensures its speedy detection, and compels everyone to seek the protection afforded by a respirator. This is in marked contrast with the insidious character of mustard gas vapour, in which the absence of immediate irritation may readily disguise its dangerous qualities.

The early detection of lewisite vapour should ensure, by immediate use of the respirator, immunity for the eyes and the respiratory tract; in the absence of suitable protective clothing, however, the skin of the body is liable to suffer through the action of the vapour absorbed by the garments, if the latter be dry. Wet clothing is a partial safeguard against the vapour, as the moisture in the material will tend to hydrolyse it.

A minor, though possibly alarming, peculiarity of lewisite vapour (which it shares with other arsenical ' gases ') is the temporary increase in the intensity of the nasal and respiratory irritation which appears *after* adjustment of the respirator, and which may lead to a lack of confidence in the respirator.

The types of injury to which lewisite vapour might give rise are summarised below.

(1) *Action on the Eyes.*

The irritancy of lewisite vapour and the consequent immediate adoption of protective measures tend to minimise effects on the eye, while the lachrymation which it induces will form an additional factor of safety; it is not likely, therefore, that lewisite vapour will produce a large number of eye casualties.

If severe burning should occur it would be analogous to that produced by mustard gas vapour, but much more acute. Under these circumstances the eye lesion will undoubtedly be accompanied by pulmonary injury.

(2) *Action on the Respiratory Tract.*

The rapid adjustment of the respirator, enforced by the intolerable character of lewisite vapour, affords the greatest security against serious respiratory lesions. In the absence of such protection, the train of symptoms is much more acute than with mustard gas vapour, for even with low concentrations the naso-pharynx is affected within a few minutes, and symptoms of coryza, with salivation and laryngeal irritation, quickly supervene. These are followed by a generalised bronchitis which is well established in 24 hours, and, which, in a severe case, may lead to broncho-pneumonia and death.

(3) *Action on the Skin.*

Wet clothing is somewhat of a safeguard against lewisite vapour, but its absorption by dry clothing renders the latter dangerous for continued wear.

Unless in high concentration on a hot, receptive skin, or on prolonged exposure (as when wearing contaminated clothing), the action of lewisite vapour does not go beyond an erythematous condition of the area affected. When vesication occurs, the skin presents

an angry surface interspersed with small, shallow, turbid blisters which may coalesce to form one large vesicle; the irritation is more marked than with the corresponding mustard gas vapour burn.

42. Nature of Casualties from Liquid Lewisite

The immediate dangers incurred by moderate or severe contaminations with liquid lewisite are similar in nature to those following contamination by liquid mustard gas: the main difference lies in the greater rapidity of action of lewisite.

The types of injury which might result are summarised below.

(1) *Skin Burns due to Liquid Lewisite.*

(a) *On bare skin.*

As with mustard gas, liquid lewisite attacks the bare skin on contact, but penetration is much more rapid. Unlike mustard gas, however, which does not produce any sensory irritation of the skin on contact, the application of liquid lewisite is usually followed by a stinging sensation which may persist for some time, and which may well prove severe if an extensive area or a hypersensitive surface be affected.

The extent and severity of the resulting damage naturally varies, as in the case of mustard gas, with the degree of contamination and the part of the body affected; experimental evidence shows, however, that very small drops of lewisite are (except in the case of the eyes) less effective than similar sized drops of mustard gas, while the reverse is the case with large drops on the bare skin or on the outer surface of dry clothing.

Erythema of the skin following liquid lewisite contamination develops rapidly—usually in 15 to 30 minutes—and, apart from this rapid development, it may be indistinguishable from that caused by liquid

mustard gas. Vesication is correspondingly early and is fully developed within 12 hours or less; it is at this stage that a clear distinction can usually be made between lewisite and mustard gas burns. The *lewisite blister* is more sharply defined, overlies practically the whole of the erythematous area, and is filled with an opaque or opalescent fluid which, on examination, is found to be rich in leucocytes and contains traces of arsenic; the typical *mustard gas blister,* on the other hand, is surrounded by an angry zone of erythema, and contains a clear, limpid, lemon coloured serum with no trace of the vesicant.

In the absence of secondary infection the healing of small, localised lewisite blisters takes place more readily than in the case of similar mustard gas vesicles; if sepsis be present, however, and contamination be widespread, it is not likely that any appreciable difference in the period of invalidism will result.

The risk of serious arsenical poisoning following liquid contamination of the bare skin has not, naturally, been studied on the human subject; the danger, however, appears to be remote unless the contamination be massive, such as might follow an accident in factories or the bursting of a lewisite bomb in close proximity to some person.

(b) *On clothed skin.*

The effects of liquid lewisite on the skin through clothing, when compared with those produced by liquid mustard gas under the same conditions, are largely governed by the degree of contamination sustained and the amount of moisture present in the garments.

Unless the contamination be massive, or the clothing thin and scanty (as in the tropics), the action of liquid lewisite on the clothed skin may be ascribed entirely to that of the vapour evolved from the contaminated area.

(2) *Eye Burns due to Liquid Lewisite.*

The dangerous possibilities of aircraft spray, already referred to in connection with eye contamination by liquid mustard gas, are intensified in the case of liquid lewisite. However small the contamination, the impact of liquid lewisite on the conjunctiva elicits immediate pain, spasm and lachrymation. The vesicant acts, subjectively, like a powerful caustic, and the victim becomes an immediate casualty; this is in marked contrast with liquid mustard gas which does not usually incapacitate until about one hour after contamination.

Clinical signs following lewisite contamination of the eye develop rapidly—much more rapidly than with similar mustard gas contaminations. An acute inflammatory condition of the conjunctiva, with pronounced engorgement of the blood vessels, is well established in 10 to 15 minutes; oedema of the eyelids, intra-ocular pain and photophobia follow rapidly, and within three or four hours the clinical picture is alarming—an intense oedema, with eyelids closed and adhering by their margins, separation of which releases purulent fluid; chemosis, submucous haemorrhages and extensive ulceration of the conjunctiva; a hazy cornea, and generally, a condition even more distressing than that produced by liquid mustard gas in 24 hours.

The prognosis in liquid lewisite contamination of the eye is even more serious than with liquid mustard gas.

43. Preventive Treatment in case of Lewisite Contamination

The extreme rapidity with which liquid lewisite penetrates the skin limits the value of all preventive treatment unless it be immediate; with lewisite, even more than with mustard gas, speed is of the utmost importance.

Theoretically, lewisite may be neutralised locally by the application of water, especially if the latter be hot and contain an alkali; it may also be removed by solvents, or it may be destroyed by bleaching powder, as in the case of mustard gas. Practically, it has been found that none of these methods is successful in preventing a burn unless applied within one minute after liquid contamination of the skin.

The prompt application of aqueous bleach or of solvents, especially if the liquid contamination be still visible on the skin, may succeed in preventing vesication; penetration, however, is so rapid that the use of bleach, if belated, may only serve to irritate an area already damaged.

This does not necessarily imply that no action can be taken to lessen the eventual consequences of contamination. The speedy removal of all contaminated clothing, followed by a hot bath combined with the liberal use of soap, is just as helpful in reducing the severity of possible lewisite burns as in the case of mustard gas, and is just as essential if further contamination is to be avoided; indeed, the period during which contaminated clothing may safely be worn is even shorter with lewisite than with mustard gas, especially if the contamination consist of large drops or splashes of the liquid.

After *vapour* exposures, a hot bath will usually suffice to ward off serious effects; irritation of the eyes or of the naso-pharynx may be treated with irrigations of sodium bicarbonate.

44. Curative Treatment for Lewisite Casualties

The curative treatment of lewisite lesions is mainly symptomatic, and follows the same lines as the treatment of mustard gas casualties. The preliminary bath advocated in the treatment of mustard gas casualties must similarly be administered to all lewisite casualties; in addition to removing all traces of the vapour from

the surface, the bath will also reduce the risk of secondary infection of any skin lesion that may subsequently develop.

If vesication occur, and especially if the area thus affected be extensive, it is essential that the fluid in the blister be evacuated early, the epithelium removed and the raw surface irrigated, in order to lessen the danger of absorption of arsenic. In the absence of sepsis such burns heal more rapidly than the corresponding mustard gas burns.

For reasons already stated, burns of the eye by lewisite vapour are not likely to be so numerous or so severe as with mustard gas; liquid lewisite contamination, on the other hand, will present a serious problem, and treatment will be difficult to carry out owing to immediate spasm and the early and extensive swelling.

Clinical evidence of arsenical absorption, as the result of gross liquid contamination of the skin, may be found in the presence of arsenic in the urine, with, possibly, signs of renal and hepatic congestion and gastric and intestinal disturbances.

CHAPTER VIII

PARALYSANT GASES

This chapter deals with two gases, hydrocyanic acid and hydrogen sulphide, which are not ordinarily regarded as war gases because, though they are extremely lethal, it is not easy to create lethal concentrations of them under war conditions. It is, however, possible that means might be devised of using them. They are also used industrially, and a description of their characteristics and effects is therefore included in this book. All respirators provided by the Government will give adequate protection against any concentration likely to be met as a result of their use in war.

45. Hydrocyanic Acid (Prussic Acid)

Physical and chemical characteristics.

Hydrocyanic acid is a clear, colourless liquid of low boiling point (26° C. or 78·8° F.), very volatile and smelling strongly of bitter almonds.* It is very soluble in water and in alcohol, but such solutions decompose rapidly. Watery solutions do not redden litmus paper.

The vapour of hydrocyanic acid is somewhat lighter than air and diffuses rapidly when released. In closed spaces it is extremely toxic; in the open, however, the dispersion of the gas is so rapid that relatively low concentrations result which are not lethal. This fact explains the failure of hydrocyanic acid gas shells in the last war in the open field, where they caused but few casualties.

Mode of action.

The gas is a protoplasmic poison which arrests the activity of all forms of living matter by inhibiting oxidation. In high concentration, such as may be

* Some persons, as an idiosyncracy, are unable to distinguish the smell of hydrocyanic acid.

found in a confined space, this gas may well be considered a fulminant poison, as it may cause death with dramatic rapidity through paralysis of the respiratory centre in the brain.

In the open, however, the gas loses much of its potential activity through diffusion; moreover, in low concentrations, the gas may be detoxicated in the body, as quickly as it is absorbed, to products which are relatively harmless. The failure of this gas in the open is therefore not surprising.

A valuable test for confined spaces is to employ susceptible animals (such as canaries, pigeons or dogs) to indicate the presence of the gas; canaries are particularly suspectible, as they succumb in about two minutes when exposed to a concentration which is not rapidly harmful to man.

Hydrocyanic acid gas may in certain circumstances be absorbed by the skin. Owing to the ease with which the gas dissolves in water, the skin absorption danger is greatly increased if the weather be hot, the cutaneous blood vessels dilated, and the skin bathed in sweat.

Symptoms.

With high concentrations the effects are rapid. The symptoms are ushered in by uneasiness and vertigo, palpitation and hurried breathing; unconsciousness and convulsions follow quickly, and death occurs through paralysis of the respiratory centre and failure of the circulation.

Concentrations that are not lethal may yet produce headache or giddiness, and sometimes nausea or inability to concentrate; recovery, however, is usually rapid and complete.

Treatment.

Treatment must be immediate, and the primary, urgent necessity, in order to restore respiration, is to reduce the concentration of the gas in the circulation.

At once remove the victim from the poisonous atmosphere, and use artificial respiration. The administration of oxygen with an admixture of 5 to 7 per cent. carbon dioxide is useful. The carbon dioxide, through central stimulation, will help to secure thorough ventilation of the lungs.

Various prophylactic and antidotal methods have been suggested based on the laboratory neutralisation of hydrocyanic acid gas by certain chemicals, such as sodium thiosulphate, methylene blue, glucose, etc., but only partial success has, so far, followed their adoption. The main essential in successful treatment is immediate artificial respiration.

46. Hydrogen Sulphide (Sulphuretted Hydrogen)

Physical and chemical characteristics.

Hydrogen sulphide is a colourless gas possessing a foetid odour resembling that of rotten eggs, more offensive in weak than in strong concentrations. Although this characteristic odour can be detected in concentrations low enough to be harmless, fatigue of the sense of smell occurs early, and the odour may cease to serve as a warning. Again, very high concentrations, though highly irritating to the eyes and throat, may be unrecognisable by the sense of smell and may, like hydrocyanic acid gas, be rapidly fatal. The gas, which is inflammable, is heavier than air. Hydrogen sulphide was tried in the last war as an offensive gas, but its use was abandoned.

Its presence may be readily detected, apart from the smell, by exposing lead acetate paper, sheet copper or slightly moistened silver articles, which are all blackened in the presence of hydrogen sulphide.

Mode of action.

Hydrogen sulphide acts both as a local irritant and as a systemic poison. Local irritation is confined to the tissues and exposed mucous membranes of the

eyes, throat and respiratory tract, while systemic poisoning follows the invasion of the lungs by moderate or high concentrations of the gas.

High concentrations of the gas produce death with the same dramatic suddenness as with hydrocyanic acid, due in both cases to paralysis of the respiratory centre in the brain. Moderate concentrations give rise to bronchitis and symptoms of pulmonary oedema. There is no evidence that abnormal combinations with haemoglobin are formed. The respirator affords protection against this gas.

Symptoms.

Symptoms of acute poisoning are usually ushered in by panting respiration, pallor and rapid unconsciousness; this is quickly followed by cessation of breathing, often accompanied by convulsive movements. The heart continues to beat for some minutes, but, unless the victim is removed from the gassed area and artificial respiration employed immediately, cardiac failure and death will result. In less acute cases of poisoning violent irritation of the eyes and severe inflammation of the respiratory tract, which may prove fatal, are the most prominent symptoms.

Sub-acute cases of poisoning, as met with occasionally in industrial life, are not likely to be seen frequently in war time; they give rise to symptoms of general ill-health, with chronic conjunctivitis and affections of the respiratory and digestive tracts.

Treatment.

As in the case of poisoning by hydrocyanic acid gas, treatment must be prompt. It consists essentially in removal of the victim from the poisonous atmosphere and the administration of artificial respiration, preferably with inhalation of oxygen mixed with 5 to 7 per cent. of carbon dioxide. The latter stimulates the

respiratory centre in the brain, and the rapid oxidation of the residual hydrogen sulphide in the blood ensures that nc lasting after-effects follow the exposure.

Artificial respiration should be persisted in for a long period, even though there may be no signs of life. This is proved by industrial practice.

The treatment of the sub-acute type is symptomatic; recovery is usually complete if permanent freedom from further exposure can be secured.

CHAPTER IX

OTHER DANGEROUS GASES AND FUMES

The gases dealt with in this chapter are not likely to be used as war gases, but may be met with under certain conditions in war, as well as in time of peace. Some of them are toxic gases, such as carbon monoxide and the nitrous fumes which are generated from burning explosives, while others are substances used for the production of screening smokes, which may be dangerous to handle. In addition, reference is made to the atmospheric dangers encountered in fighting fires.

47. Carbon Monoxide

Carbon monoxide is a colourless, odourless gas, extremely insidious because of its entirely non-irritating character, and the consequent impossibility of recognising its presence in the atmosphere by the senses. It always accompanies any process of incomplete combustion of carbonaceous material, and is therefore commonly met under normal conditions of everyday life. The gas burns with the characteristic blue flame so often seen flickering over a coke or smouldering coal fire, and it forms an explosive mixture with air.

Carbon monoxide is always present in dangerous amounts in the exhaust gases of internal combustion engines, coke stoves or smouldering fires, while varying quantities of it are present in all types of illuminating gas; it also forms the deadly constituent in the so-called " after-damp " in mines.

Carbon monoxide may be met with in dangerous quantities as a result of bursts of high explosive bombs, in blasting operations, and in burning buildings, if the conditions confine the fumes in a narrow space, as well as in the event of fracture of gas mains and pipes.

The respirator does not afford any protection against carbon monoxide, but complete protection is of course given by a self-contained oxygen breathing apparatus, such as is used by firemen.

Small animals, such as mice or canaries, can serve as valuable indicators of the presence of carbon monoxide as, owing to their rapid metabolism, they show signs of carbon monoxide poisoning before man is affected.

Mode of action.

Carbon monoxide owes its poisonous properties to the fact that it combines with haemoglobin to form a dissociable red compound just as oxygen does, and that its affinity for haemoglobin is about three hundred times that of oxygen. But for this property of combining with haemoglobin carbon monoxide would be a physiologically inert gas like nitrogen or hydrogen.

When a man breathes air containing carbon monoxide, the relative amounts of oxygen and carbon monoxide present in the atmosphere determine the proportion in which carboxyhaemoglobin is found in the blood. When the amount of oxygen is three hundred times that of carbon monoxide half of the haemoglobin can combine with the carbon monoxide and half with the oxygen. This is about the blood-saturation at which unconsciousness occurs.

As the concentration of the gas in the air rises, the saturation of the haemoglobin with carbon monoxide increases, and the oxygen-carrying capacity of the blood progressively diminishes until symptoms of anoxaemia (oxygen want, in this instance without cyanosis) make their appearance. Carbon monoxide is therefore cumulative in its action.

Moreover, the rate of absorption of carbon monoxide is very much accelerated by muscular exertion or by mental excitement, which causes an increase in the breathing and circulation rates. This results in a more

rapid diminution in the available oxygen content of the blood, with a corresponding increase in the severity of the symptoms of oxygen want.

The resulting effects are due to anoxaemia alone, and their severity is determined by the degree of oxygen want. There are no pathological changes in the lungs such as follow the inhalation of asphyxiant gases, nor are the red blood corpuscles injured; when freed from their combination with carbon monoxide, the corpuscles are as capable of resuming their normal function as oxygen carriers as they were before exposure to the gas.

Death occurs when saturation of the haemoglobin reaches about 75 per cent., but lower degrees of saturation of the blood may prove fatal if exposure to the poisonous atmosphere is prolonged. The colour of the blood and tissues, *post mortem,* may be bright red.

Symptoms.

The great danger in carbon monoxide poisoning is that, owing to the non-irritant properties and the cumulative action of the gas, the victim may not realise, until too late, that there is any danger present in the atmosphere. The first symptom may be a loss of power in the limbs so that, although he may then appreciate his danger, escape may be difficult or impossible.

Where the proportion of carbon monoxide to oxygen is high, loss of consciousness may be very rapid, with practically no warning. More commonly, however, the onset of symptoms is gradual and insidious, and may be ushered in by a feeling of weakness, giddiness, vomiting, and indistinct vision; this is followed by breathlessness, palpitation and a loss of power in the limbs, and the least exertion at this stage may cause collapse.

The loss of muscular power and the confused cerebration often preclude a man from withdrawing from danger even though he is dimly aware that safety is

only a few yards distant. Not infrequently there is a stage of acute mental excitement, which may simulate alcoholic intoxication or even mania. This is more common in the milder cases. Apathy and a sense of complete helplessness supervene, followed by unconsciousness, with or without convulsions; the victim becomes comatose, with stertorous breathing, a low-tension pulse and subnormal temperature, and death results if he is left in the poisoned atmosphere.

The colour of the face in cases of carbon monoxide poisoning may vary with the rapidity of the onset or the degree of anoxaemia. A leaden tint is often seen after profound coma, while in other cases the face may be pale and moist with perspiration; often, however, the cheeks are pink and the lips of a vivid carmine tint.

At the autopsy, the blood may be red in colour instead of dark if there is a considerable degree of saturation of the haemoglobin with carbon monoxide. If the case has continued to breathe for some time after reaching an atmosphere free from carbon monoxide this gas will have been partly or entirely displaced from the haemoglobin, and the blood after death will have its normal colour.

The simplest method of detecting the presence of carbon monoxide in blood is to compare the colour of a dilute solution of the suspected blood with a similar solution of normal blood. Take a drop or two of blood from the finger of a normal person and dilute it in a test tube very considerably with water (a $\frac{1}{2}$ per cent. solution is a convenient strength) so that when examined by transmitted daylight the colour of this solution is a reddish yellow. Then take a drop or two of the suspected blood and dilute it similarly with water so that the depth of colour of the solution is the same as that of the solution of normal blood when both are viewed by transmitted light. On examining the quality of the colour it will be

found that the solution made with the suspected blood, if it contains carbon monoxide haemoglobin, is definitely pinker than that made with the normal blood; though it will not have the full pink tint of the same normal blood solution if the latter be shaken with coal gas so as to saturate it quite completely with carbon monoxide.

Recovery from the initial symptoms may be followed by some degree of mental confusion and slow cerebration which may persist for varying periods, while headaches, often of a severe or migrainous type, are characteristic. Among the after-effects of severe poisoning may be mentioned cardio-vascular disorders, especially tachycardia and dyspnoea, which may continue for months. There is a predisposition to pneumonia, usually as a sequel to a long exposure to the gas, while disturbances of the central nervous system may occur ranging from a simple neuritis to paresis and even mental derangement, usually of temporary duration, though sometimes lasting as the result of cellular damage to the brain caused by protracted anoxaemia.

Treatment.

The majority of cases of carbon monoxide poisoning recover with prompt treatment, although a relapse, or even sudden death, may occur where exposure to the gas has been prolonged.

The time required for recovery depends however on how long the victim has been exposed to the poisonous atmosphere. Unconsciousness may last for as long as 48 hours after regaining pure air, and yet the person may recover; but the longer unconsciousness lasts the less is the chance of recovery.

Treatment consists in the prompt administration of oxygen, or *preferably* oxygen, or even air, *combined with 5 to 7 per cent. of carbon dioxide,* aided, if necessary, by artificial respiration.

Pure oxygen can displace the carbon monoxide far more rapidly than ordinary air, while the carbon dioxide stimulates the respiratory centre and induces deeper breathing, thus facilitating the elimination of carbon monoxide from the circulation. It is hardly necessary to add that the expired air must not be re-breathed by the patient, and with this in view, a suitable apparatus should be used such as the Haldane oxygen apparatus, which may be set to deliver eight to ten litres a minute, or one of the types of apparatus which allow the administration of a mixture of carbon dioxide with either oxygen or with air.

A characteristic effect of carbon monoxide poisoning is a lowering of the body temperature, due to a disturbance of the heat regulating centre and to a reduction in the normal oxidative processes. Even in mild cases patients may complain bitterly of cold, and it is necessary that this symptom be combated by means of hot coffee, blankets, hot water bottles and other familiar measures. Rest, too, is imperative in order to avoid any increase in the oxygen requirements of the body and to reduce the demands on the ill-nourished heart.

In serious cases a slow intravenous injection of 5 ccs. of 25 per cent. coramine solution, or similar respiratory stimulant, is valuable. Blood transfusion has been found of great value in desperate cases. Venesection is quite valueless.

During convalescence, especially after severe or prolonged anoxaemia, particular care should be taken that no great strain be thrown on the heart owing to the risk of acute dilatation.

48. Nitrous Fumes

When nitro-explosives are incompletely detonated or subjected to slow combustion, especially in confined spaces, considerable quantities of nitrous fumes are given off consisting chiefly of nitric oxide (NO) and nitrogen peroxide (NO_2 and N_2O_4).

These fumes, which have an orange-yellow or reddish-brown colour, are very soluble in water, and react readily with moisture and oxygen to form nitric and nitrous acids. In damp surroundings, therefore, the concentration of these gases in the atmosphere will be lowered.

Nitrous fumes may be met with when detonation of a blasting charge is incomplete. In industry, dangerous concentrations may be evolved when nitric acid is heated, or when it comes in contact with organic material, such as wooden floors, after accidental spilling.

The respirator affords limited protection against nitrous fumes, but as it gives no protection against carbon monoxide, which is often generated simultaneously, reliance should not be placed on a respirator save in an emergency.

Nitrous fumes may be readily demonstrated by means of test papers which have been previously dipped in a solution of starch and potassium iodide and slightly acidified; a blue coloration develops on them when they are exposed to the gases.

Though even small amounts of nitrous fumes in the air are mildly irritating to the eyes and the respiratory tract, they are not sufficiently so to serve as a warning of a highly dangerous atmosphere.

Mode of action.

The action of nitrous fumes on the lungs closely resembles that of phosgene. They are particularly dangerous because they do not produce any marked sensory irritation, and men may therefore fail to realise the serious danger which may follow their inhalation.

When inhaled, the nitrous fumes come into contact with the moisture ever present in the respiratory tract, and form nitric and nitrous acids; this produces a local caustic effect, to which is superadded a general systemic

action due to absorption of the alkaline nitrites formed by the interaction of the acids with the alkaline secretions in the presence of oxygen.

As with phosgene, the local action gives rise to an intense congestion of the lungs and the production of an acute inflammatory condition and pulmonary oedema. This always overshadows the general systemic effect of the alkaline nitrites, which, however, contribute to the clinical picture through their enfeebling action on the circulation and the possible diminution, through the formation of methaemoglobin, of the oxygen carrying capacity of the blood.

With nitrous fumes, as with phosgene, the initial symptoms of coughing and irritation are generally transitory, and a period of quiescence precedes the onset of the acute symptoms. This apparently delayed action may vary in duration from two to 24 hours or more, according to the conditions of exposure; the usual duration of the period is between 10 and 20 hours after exposure. Once this period is over, the clinical signs develop rapidly, and the whole course of a possibly fatal illness may be run in a few hours.

Symptoms.

The initial symptoms, on exposure, are slight irritation of the eyes, nose and throat, accompanied perhaps by a little cough—symptoms which are seldom marked and which quickly subside during the latent period which follows. The termination of this latent period, which may be precipitated by physical exertion, is marked by the onset of acute clinical signs and symptoms such as a dry hacking and painful cough, a sense of constriction in the chest, and distressing breathlessness.

In mild cases this may be a prelude to a bronchitis which is limited to the upper bronchi and is accompanied by a profuse muco-purulent expectoration. In more severe cases, however, a condition of acute bronchial spasm may set in. with pulmonary congestion

and cyanosis, rapidly followed by a pulmonary oedema which may be haemorrhagic in character. Restlessness is extreme, and in fatal cases consciousness is retained almost to the end, the patient struggling vainly for breath while, with blood stained fluid trickling from his mouth and nostrils, he drowns slowly in the fluid exuded in his lungs.

Treatment.

The general principles of treatment in cases of poisoning by nitrous fumes follow the same lines as those already outlined for cases of phosgene poisoning, stress again being laid on the importance of enforcing complete physical rest from the time of exposure and on the early administration of oxygen as soon as cyanosis develops.

Venesection gives better results when practised early, even before the onset of pulmonary oedema, in subjects with a full bounding pulse, but it is generally contra-indicated where the pulse is soft and thready.

It should be remembered that a severe and often fatal broncho-pneumonia is a complication in some cases of lung irritant poisoning.

Convalescence is apt to be prolonged, and the experience of the last war showed that the combined action of carbon monoxide and nitrous fumes had a harmful effect on the heart, necessitating careful surveillance and graduated exercises.

49. Screening Smokes in General

The five substances described in the next five sections can be used as screening smokes to conceal important places. These smokes are irritating when inhaled in close proximity to their source, but are not toxic in the concentrations that render them effective as screens.

The chief danger associated with their use arises through accidental contact with the chemicals used in their production. These chemicals are all corrosive or dangerous to handle, and accidental contamination of the eye or splashes on the skin with the liquids will result in severe ulceration or burns.

Should contamination with the liquid chemicals occur, first aid treatment must be undertaken immediately. If the eye be affected, prompt and copious lavage with water, or with warm sodium bicarbonate solution, may mitigate the resulting effects; splashes on the skin should be treated with excess of water in order to dilute rapidly and wash away the corrosive liquid, while any article of clothing contaminated by the chemical should be discarded at once.

The respirator gives efficient protection against all the screening smokes, and clothing is not affected by exposure to them in the concentrations met in the open.

50. Phosphorus

At ordinary temperatures phosphorus is a solid which can be handled safely in water, but when dried in air it burns fiercely, producing a dense white smoke.

Phosphorus may also be used by an enemy as an incendiary filling in bombs or in shell, and flying fragments or melted particles of the burning chemical may be embedded in the skin of persons close to the bursting missile. These fragments continue to burn on the skin unless smothered; first aid treatment should therefore consist in the immersion of the affected part in water, or, in the absence of enough water, in the application of a thick pad soaked in water.

As the melting point of phosphorus is 44·4° C. (112° F.), the particles embedded in the skin can be removed, under water at or above this temperature, by means of forceps or a gauze sponge, care being taken that none of the fragments be overlooked.

The resulting burns should be treated as thermal burns, but they are apt to be slow in healing. Owing to the ready solubility of phosphorus in oils and fats, no oily or greasy dressings should be used until it is certain that all the fragments have been removed.

A form of first aid advocated by American writers is the immersion of the affected part in, or the covering of the area with, thick pads soaked in a 1 or 2 per cent. solution of copper sulphate. The action is to coat the particles of phosphorus with an inert compound by chemical action, and so to arrest the burning and enable removal to be carried out.

51. Chlorosulphonic Acid (C.S.A.)

This is a fuming, highly corrosive liquid which, on contact with quicklime, gives off a thick white cloud closely resembling a dense mist. At close quarters this is sufficiently irritating to the eyes and throat to necessitate the wearing of a respirator, but at a distance of 200 yards or more from the source of emission this can easily be dispensed with.

Owing to its highly corrosive nature, C.S.A. requires great care in handling; moreover, in contact with water it generates intense heat and acid may be scattered in all directions.

Contamination of the eye with the liquid should be treated immediately with large quantities of water, followed by lavage with a 3 per cent. sodium bicarbonate solution; a few drops of castor oil and a light pad over the eye will assist in allaying the irritation.

Splashes on the skin should be flooded with water to remove the contaminant, and sodium bicarbonate solution should be applied locally thereafter. Drying should be effected by mopping up excess moisture gently with swabs or absorbent wool, and not by rubbing.

52. Oleum

Oleum is a brownish-yellow, corrosive liquid consisting of sulphuric acid with a percentage of sulphur trioxide; when exposed to air it gives off dense white fumes with a somewhat sulphurous smell.

As with C.S.A., accidental splashes should be treated immediately with an excess of water to wash off the acid, followed by the local application of sodium bicarbonate solution.

53. Titanium Tetrachloride

This is a yellow, non-inflammable and corrosive fluid which, on contact with damp air, gives off a heavy dense white cloud. Advantage is taken of this property for the production, by aircraft, of vertical smoke curtains extending down to the ground or sea level. The smoke consists mainly of fine particles of free hydrochloric acid and titanium oxychloride, and its efficiency depends largely on the moisture present in the air.

The smoke is unpleasant to breathe, but it is not toxic; the wearing of goggles or a respirator, however, may be necessary when entering a smoke curtain if the spray is still falling, owing to the danger of drops entering the eyes. The usual precautions must be taken when handling the liquid, and accidental contamination of the eyes or of the skin should be treated immediately by free lavage with water, followed by local application of sodium bicarbonate solution.

54. Stannic Chloride

Stannic chloride is a fuming, straw-coloured, corrosive liquid which produces a heavy white cloud on contact with air, and is therefore sometimes utilised by aircraft for the production of vertical smoke curtains. The treatment necessitated by accidental splashes is similar to that associated with titanium tetrachloride.

55. Noxious Products of Combustion

It is common knowledge that fire fighting, especially when practised inside buildings or in confined spaces, often entails risk, either because of noxious gases or through a deficiency of oxygen; hence the various protective devices used by firemen such as respirators and oxygen breathing apparatus.

In all fires in confined spaces the nature and concentration of the toxic gases produced vary with the rate of combustion and with the character of the burning material. Thus, a slow rate of combustion results in a heavy concentration of carbon monoxide and carbon dioxide, in addition to an oxygen deficiency, while burning cordite (as in a magazine) gives rise, in addition, to the evolution of nitrous fumes.

When chemical extinguishers are used to quell fires in confined spaces, additional toxic gases may be produced causing further danger to unprotected men.

The unfailing presence of carbon dioxide hastens the onset and increases the severity of any toxic symptoms that may result. Carbon dioxide is more than a simple asphyxiant in that, in comparatively low concentrations, it causes increased breathing and thereby increases the quantity or dose inhaled of any noxious gas that may be present. In concentrations above 20 per cent. it produces unconsciousness and death.

The utility of the ordinary anti-gas respirator in fire fighting is strictly limited, and is confined to the arrest of particulate products of combustion and of such gases as can be dealt with by the charcoal in the container. These do not include either carbon monoxide or carbon dioxide. Further, as the respirator cannot compensate for any oxygen deficiency that may arise, it is essential that, when fighting fires inside confined spaces where a free dilution of the atmosphere or a free escape for the noxious gases generated is not possible, an oxygen or air breathing apparatus should be worn.

The toxic properties and mode of action of carbon monoxide and of the nitrous fumes have been described. A short description is appended below of the toxic effects of two types of fire extinguishers in common use, followed by a brief study of the conditions under which a deficiency of oxygen may be met.

56. Dangers from Use of Chemical Fire Extinguishers

The following are two types of filling for chemical fire extinguishers in common use, sold under various trade names:—

(1) *Carbon Tetrachloride.*—This is a volatile liquid, boiling at 76·7° C. (170·1° F.), which is extensively employed as a dry-cleaning agent and as a popular and effective fire extinguisher.

When carbon tetrachloride is sprayed on a fire or on a heated surface the chief decomposition products, in addition to the unchanged chemical, are phosgene, hydrochloric acid and chlorine. The production of phosgene is more marked when the liquid comes in contact with heated rusty iron and when large quantities of the extinguisher are used in the presence of moisture.

Although the thermal decomposition products are more or less irritant to breathe, this irritancy may not be such as to compel men, faced with a dangerous emergency, to leave a burning room. Under these circumstances a very real danger arises from the continued inhalation of vaporised carbon tetrachloride or its products of decomposition.

Recent experience has shown that exposure to the fumes of carbon tetrachloride itself in a confined space such as a garage or between decks may give rise to serious illness, often delayed in its onset, of renal and hepatic origin. The illness may be ushered in by pyrexia, general malaise and abdominal pain—a commonplace clinical picture which may lead to errors in diagnosis.

Personal idiosyncracy plays a part in the character, as well as in the severity, of the resulting symptoms; but as a rule, signs of impaired kidney function are always present, and may vary from a trivial rise in blood pressure to an acute uraemia. Evidence of liver damage may also be seen in the jaundice, the slow pulse, the abdominal pain and haemorrhage from stomach and bowel so characteristic of the toxic jaundice caused by the organic halogens.

(2) *Methyl Bromide.*—Another type of fire extinguisher contains methyl bromide as its chief constituent. This is a gas at ordinary temperatures, but it is readily liquefied at 0° C. to a clear, colourless and extremely volatile liquid which boils at 4·5° C. (40·1° F.). The gas is almost odourless.

Methyl bromide is toxic, and its thermal decomposition products are practically irrespirable. The liability of this extinguisher to produce poisoning, however, is chiefly determined by the rapid rate of volatility of the undecomposed chemical, which is much higher than that of carbon tetrachloride. The rapid vaporisation of methyl bromide in confined spaces may easily result in such a high concentration that a toxic dose may be inhaled before the danger is appreciated.

In high concentration methyl bromide has a profound effect on the central nervous system, producing unconsciousness and giving rise to epileptiform seizures and paralysis, both motor and sensory.

In less severe cases vertigo, visual troubles and general weakness are the usual symptoms, and it appears that the dose need not be large to produce such symptoms.

57. Dangers of Oxygen Deficiency

Oxygen deficiency occurs as the result of fires in confined spaces, and the presence of carbon monoxide and carbon dioxide adds to the danger.

There are many situations, however, other than actual fires in closed compartments, where a reduction of the oxygen percentage in the atmosphere may occur. Apart from such occasions as high altitude flying or climbing, oxygen deficiency may be met with, both in peace time and under war conditions, under the following circumstances:—

(*a*) In the air of wells, disused mine galleries, underground shafts and tunnels, etc. Through the oxidation of organic and mineral matter in the soil, the composition of the air in such spaces may be seriously, and sometimes totally, deficient in oxygen on first entry. Even after thorough ventilation a constant watch on the purity of the atmosphere in the space must be maintained, as a fall in the barometric pressure tends to fill the confined area with residual nitrogen welling out of the surrounding strata.

(*b*) In air-tight compartments such as double-bottoms of ships. When these compartments are sealed for any length of time, the whole of the oxygen in the enclosed air may be used up by the ordinary process of the rusting of iron or steel bulkheads or through oxidation of the linseed oil in the paint commonly used in these spaces.

(*c*) In badly ventilated compartments, such as ships' holds or coal bunkers, in which oxidisable or oxygen-absorbing substances, such as grain, fruit, potatoes or coal, are stored.

Symptoms of anoxaemia.

In healthy adults, the percentage of oxygen in the air breathed must be reduced by about a third before any symptoms become obvious, unless heavy muscular work is being undertaken. If exposure to such an atmosphere be maintained, or if the concentration of oxygen be reduced still further, a chain of symptoms follows which is insidious in its onset and which is typical of anoxaemia from whatever cause it may arise,

namely:—headache, visual disturbance and mental dullness, loss of muscular power and of co-ordination, dyspnoea and weakened cardiac action; the power of judgment may be seriously impaired, while loss of memory is of common occurrence. There may be progressive cyanosis of the grey type with circulatory failure. With an extreme reduction in the oxygen percentage in the atmosphere, a condition of acute anoxaemia results in immediate loss of consciousness.

Treatment.

Immediate removal to fresh air is imperative, and, if the breathing has stopped, artificial respiration should be resorted to at once. This should be supplemented, if possible, by the administration of oxygen, by means of the Haldane oxygen apparatus at the rate of 8 to 10 litres per minute; the addition of 5 to 7 per cent. of carbon dioxide to the oxygen will greatly enhance the value of the latter by stimulating the respiration.

Precautions.

The precautions which may be taken may be summarised as follows:—

(*a*) Test the suspected atmosphere by means of a safety lamp, such as is commonly used in coal mines, for fear of the possible presence of explosive or combustible gases. A flame is very sensitive to any variation in the oxygen percentage of the air, and will be extinguished with a fall of 3 to 4 per cent. in the oxygen content at ordinary barometric pressure.

In this connection it may be useful to remember that a state of negative pressure may exist in an air-tight compartment owing to the reduction of its oxygen content, and that on opening the space the initial rush of pure air may dilute the atmosphere of the space in the immediate neighbourhood of the opening. This dilution may be

sufficient to allow a candle to burn at the entrance, but may not affect the dangerous character of the air in remote parts of the same compartment.

(*b*) Thorough ventilation of all suspected spaces or compartments, including all remote corners.

(*c*) The invariable use of a life-line attached to the body of the first person to enter the space and of any unprotected rescuing personnel.

(*d*) The employment of the only sure safeguard both against the presence of noxious gases and the absence of a sufficiency of oxygen, namely, any type of self-contained oxygen breathing apparatus.

CHAPTER X

THE RECOGNITION AND DIFFERENTIAL DIAGNOSIS OF GAS CASES

A suitable introduction to this Chapter is the following quotation from the Official History of the War, Medical Services, "Diseases of the War", Vol. II (1923), page 317. It refers to the period of the battle of Loos (September, 1915), when phosgene was being used and before mustard gas was introduced.

> "A report from the West Riding Casualty Clearing Station, which admitted 248 gas casualties, stated—'The majority of cases of gas poisoning received at this hospital showed no sign of that condition'. Other Medical Officers frankly admitted that they were so handicapped by their lack of experience of cases of gas poisoning that they were often in doubt whether they were dealing with men suffering from gas poisoning or not."

This report emphasises the difficulties of diagnosis that will always arise when persons are exposed to the dangers of gas poisoning and when neither they nor their medical attendants have had practical experience of the results of such poisoning.

58. The Importance of Accurate Diagnosis

In this chapter an attempt is made to indicate and deal with the difficulties of medical men to whom gas casualties are sent for treatment. The proper treatment of such cases demands a correct diagnosis of the condition from which they are suffering. In order to avoid men being sent back to duty when they are in the quiescent period which may follow even severe gassing with a lethal gas such as phosgene, and also to prevent the evacuation as casualties of those suffering

merely temporary discomfort from lachrymatory or sensory irritant gases, it is essential that all medical men should be familiar with the symptomatology of gas poisoning.

The doctor confronted with a supposed " gas " case is presented simultaneously with three problems. First: is the patient really suffering from the effects of gas at all, and, if so, to what extent? Second: if so suffering, then from the effects of what particular gas, or more properly from the effects of which of the recognised groups of poison gases? Third: does the case present such novel features as to suggest the use of some unknown gas?

All three questions are equally important. On the answer to the first will depend the main decisions as to the general line of management required. On the answer to the second will depend the decision as to the definite line of treatment to be adopted. If the case falls in the third category, it is essential that this vital fact should be made known immediately to those responsible for research upon the problems of treatment, of prevention, and of protection.

59. General Rules for Diagnosis

When there is doubt as to whether a person has been gassed or not, it is an important rule to withhold decision for 24 hours.

When dealing with early gas cases always give the patient the benefit of any doubt. Force is lent to this injunction by a study of the many reported cases of delayed action, and the severe and frequently fatal results of erring on the wrong side, particularly in cases due to lung irritant gas.

It is here that the value of enforced rest becomes so obvious. This period of rest, under fairly close expert

supervision, can very well be taken in many cases in the patient's own home.

If no further symptoms develop within the 24 hours, close supervision may be discontinued.

It should be clearly recognised that the known and classified groups of war gases divide themselves broadly but importantly into two main classes—(*a*) those which possess the power of acting only upon the end organs of some specific sensory nerve or nerves, or upon some special tissue, and (*b*) those which can attack any living tissue with which they come in contact.

Recognition of this fact is one of the main points of intelligent differential diagnosis. Added importance attaches to this fact because the likelihood of meeting poisons which do not fall into one or other of the recognised groups is relatively very remote.

The development of obvious features of poisoning will generally make it possible in less than 24 hours to classify the case as having resulted from either skin vesicant or pulmonary irritant gas, while further differentiation may often be indicated by information as to the type of gas that had been employed over the area in which the casualty occurred.

For the diagnosis of cases due to the recognised war gases the outline chart given in Appendix D will, it is hoped, prove of value. By the method of elimination it should enable the doctor to fit the case into one or other of the four recognised groups of Tear Gas, Nose Irritant Gas, Lung Irritant Gas, and Blister Gas, or, failing that, to label it either as not due to gas at all, or else as due to a new factor.

The term "new factor", as used above, does not necessarily imply a new chemical substance. What it does mean is that effects are being produced which do not correspond with the recognised possibilities of the materials included under the standard four-group classification.

60. Recording of Case Histories

The problem is, therefore, how existing knowledge of chemical warfare agents can be used to the best advantage?

To obtain a history either from the patient, or from others in a position to supply the details, is of the utmost importance. History, in these cases, is of far greater value and importance than in the ordinary medical or surgical case. Because of its importance it should be fully detailed, and every effort commensurate with the exigencies of the situation should be made to verify the details.

The history of the manner of development of symptoms in the first 24 hours gives the clue to the diagnosis. The progress of the case in the next few days determines the time subsequently needed for convalescence. Both must be recorded, since the casualty may later pass to other centres for treatment, and the doctors there will require accurate information as to the early stages of the cases for proper guidance in their work.

Questioning, whether of patient or of others possessing or likely to possess the required knowledge, should follow some such lines as:—

(i) When did the alleged gassing take place, e.g. minutes, hours, or days previous?

(ii) Under what conditions did the alleged exposure to the gas take place, e.g. in the open or under cover, during action or at rest, etc.?

(iii) What did the gas smell or look like?

(iv) Was any special local condition or circumstance noted, e.g. type of bombs or shells falling (gas projectiles burst with much less violence than high explosive and do not flame like incendiary); peculiar smell and, if so, how described; unusual appearance of immediate area such as might be caused by persistent gas, etc.?

(v) For how long (seconds, minutes or hours) did the exposure last?

(vi) Was a respirator available, and of what type; if so, was it put on and for how long?

(vii) Were the effects produced immediately or after some delay, and, if the latter, after how long?

(viii) What effects, if any, were produced at the time?

(ix) What effects, if any, appeared later?

(x) Did the symptoms persist after escape from the polluted area or adjustment of the respirator?

When considering the replies (and in cases of serious doubt it is as well to make a written, tabulated statement, if the conditions of the moment make this at all possible), it must be remembered that quite as much value should be given to *negative* evidence as to positive.

As examples of negative evidence might be instanced the fact that the absence of any statement relating to the air passages in a case complaining of acute and immediate eye effects would be strong presumptive evidence against a lung irritant as the probable cause, whilst the absence of any acute effect at the time of exposure in a case with obvious eye trouble would be strong presumptive evidence in favour of mustard gas.

Particular attention is directed to the answers obtained to question (v)—" how long did the exposure last? "—and it must be very clearly understood, and quite definitely conveyed to the patient, that this question links up very directly with question (vi)—" was a respirator available and, if so, was it put on and for how long? "

Furthermore, the question relates both to the period of exposure without, *and* to the period of exposure after adjustment of the respirator.

These answers may have a very direct bearing upon prognosis and treatment. For instance, if a patient alleges exposure to mustard gas vapour without a respirator for some hours and yet exhibits only slight eye and/or skin effects 24 hours later there need be no great fear of any serious lung damage.

On the other hand if the alleged exposure had taken place at night and the fact could be elicited that the patient had slept most of the time, then the effects on the closed eye might be quite mild but the damage to the skin and lungs extensive and serious.

The significance of the replies to the questions will now be considered, and the various types of gases dealt with in turn.

61. Lachrymatory Gases: Diagnosis

These gases are easily recognisable by the *immediate* and severe lachrymation and the spasm of the eyelids that they produce. As met with under war conditions they are otherwise relatively harmless, and affected persons do not usually require any treatment whatsoever. Their effects are transitory except in the unusual event of a drop of actual liquid reaching the eye, as might happen to those close to a bursting bomb or shell. The condition is not serious and clears up quickly, in sharp contrast with that resulting from eye contamination by drops of liquid vesicant.

62. Nose Irritant Gases: Diagnosis

With these gases, the severity of the symptoms which may follow within a few minutes after inhalation may mislead an inexperienced person. The subject may look very distressed, and usually complains of intense pain in the naso-pharynx, throat and chest, with aching of the gums and teeth; coughing, sneezing, profuse salivation and expectoration are generally marked, while retching, or even vomiting, may be present.

Such a patient is only too well aware that he has been acutely poisoned by gas, and may even think that he is in danger of losing his life. Paradoxically, despite the misery associated with such intense symptoms there is little serious danger associated with this type of gassing. The treatment consists of a brief period of rest coupled with the assurance that recovery will be rapid and complete.

63. Asphyxiants: Diagnosis

The asphyxiant gases—chlorine, phosgene, di-phogene and chloropicrin—have each their own distinctive smell, and the history of the patient's subjective symptoms should be of value in determining to which particular gas he was exposed. Chlorine and chloropicrin vapours are intolerable to breathe even in concentrations which are relatively harmless; their action is in no way insidious, and, to persons provided with a respirator, this quality of extreme irritation serves as a safeguard against pulmonary injury. With the vapours of phosgene and of di-phosgene, which resemble each other fairly closely, the same does not hold good. Concentrations which may be detected readily enough by their smell of mouldy hay, and by the cough and slight lachrymation that they cause, may not prove in any way intolerable to breathe, and serious or even fatal results have followed the prolonged inhalation of these gases in low and relatively non-irritant concentrations.

If there is any reason to suppose that people have been exposed to these gases without respirators they should be examined with the greatest care. A history of slight lachrymation accompanying the inhalation of an asphyxiating gas which induced coughing will usually be given. The history may also describe the smell and the choking character of the gas, the cough it produced and the tightness of the chest felt at the time; symptoms which may have been accompanied by vomiting.

The greatest difficulty may be experienced in assessing whether a person is likely to become ill as the result of such inhalation. There may be no trace of cyanosis, but rather a mild pallor and coldness of the extremities due to shock. Attacks of coughing may recur which may be accompanied by a profuse frothy expectoration, while pain in the chest, particularly on deep inspiration, may be present and respiration may be rather shallow and rapid; headache and severe general fatigue may be pronounced.

Owing to the nervous state of such persons following an attack of gas, the character of the pulse is no sure guide unless it is unusually disturbed. Examination of the chest in the early stage after the inhalation of an asphyxiant gas usually reveals little abnormality. Before the development of extensive oedema a few moist râles may be heard in the axillae or over the base of the lungs, but their absence does not justify the supposition that the lungs have escaped injury.

An examination of the blood may also prove negative, as pulmonary oedema in the early stages does not necessarily give rise to any observable change in the concentration of the peripheral blood. When oedema is fully developed, the haemoglobin may reach 120, while the red cell count of the peripheral blood may even attain the figure of 8 million per cubic millimetre. But by this time the severe clinical features of acute pulmonary oedema will suffice to give the needed information.

The conclusion which we must therefore accept is that, following exposure to a gas of the asphyxiant group which has caused definite discomfort at the time, all should remain at rest under medical care for 24 hours. The only persons who may safely be discharged are those who are known to have been exposed to low concentrations for a short period (a few minutes) only, and who show no observable residual symptoms as described above.

64. Mustard Gas Vapour: Diagnosis

The faint garlic-like odour of mustard gas vapour may readily pass unnoticed: but if it is appreciated and the respirator is adjusted immediately, there will be no ill effects to the eyes and lungs. Some two to 24 hours later, however, skin burning may develop, if the person concerned remained in the mustard gas atmosphere, in ordinary clothing, while wearing his respirator. If, therefore, conjunctivitis, laryngitis and reddening of the skin are absent some 24 hours after exposure the person concerned may be assumed to have escaped injury from the gas.

If, however, about 24 hours after exposure, and without acute onset at the time of exposure, the eyes show conjunctivitis, with some loss of voice and a general reddening of the skin, usually most marked in the genital region, it may be concluded that the patient is suffering from the effects of mustard gas vapour.

A further period of 24 hours at the most will show whether the eye condition is of sufficient severity to require hospital treatment. As has been mentioned, although the eyes are the parts of the body most vulnerable to the effects of mustard gas vapour, the pulmonary and skin damage may exceed that caused to the eyes if the patient was exposed to the vapour during sleep.

65. Arsenical Vesicant Vapour (Lewisite): Diagnosis

Exposure to lewisite vapour has not the same insidious quality as a similar exposure to mustard gas vapour. Lewisite vapour has a very strong geranium-like odour, and in a minute or two causes sharp stinging in the nose and a burning pain in the chest. The degree of lachrymation produced is mild, but the markedly hostile character of the atmosphere is so

quickly apparent to the senses that it compels those exposed to it to move out of it or to put on their respirators.

People who wear respirators in a high concentration of lewisite vapour, but are otherwise unprotected, may suffer from skin burning similar to that caused by mustard gas. These burns, however, differ from mustard gas burns in their more rapid development and the more severe irritation of the skin. As a rule there should be no difficulty in discriminating whether the vapour of mustard gas or lewisite was the causative agent in skin burns on persons who wore respirators, as, apart from any other distinctive sign, their clothing will have the strong pungent reek which is the particular characteristic of these vesicant arsenical vapours, and which cannot possibly be confused with the faint garlic-like odour of mustard gas.

66. Liquid Mustard Gas and other Vesicant Liquids: Diagnosis

Liquid vesicant contamination of the skin or clothing of persons may be visible and easily recognised; on the other hand the presence of a few stray drops on the clothing can easily escape detection. To help in the immediate recognition of liquid vesicants when sprayed from aircraft, detectors have been devised of which the simplest form is a stiff paper disc previously painted with a special yellow paint; a drop of the liquid vesicant falling on it immediately shows up as a bright red spot. Although this device is of the greatest value, it must be remembered that if the spray is sparse in its distribution—as may occur downwind of the spraying aircraft—it may miss the small area presented by a single detector disc.

The very faint smell of a few drops of mustard gas on the clothing and the lack of any skin irritation at the time give an insidious character to this form of attack, and contamination may only become evident when skin burning follows in a few hours.

The treatment required is the removal of the clothing with the least possible delay, and thorough bathing of the body.

With lewisite or other arsenical vesicants it may not be so difficult to recognise a slight liquid contamination of the clothing, as, even if the strong smell has passed unnoticed, the skin beneath the contaminated spot usually begins to sting within a few minutes. These two features should render the detection of lewisite on clothing less difficult than is the case with mustard gas.

Under certain circumstances, however, people may be sprayed so heavily that there can be no doubt about their contamination; they may be visibly covered with drops of liquid, and their skin and clothing may reek of the contaminant. The immediate cleansing treatment of such persons is not generally the responsibility of the doctor; but if the eyes have received liquid contamination, immediate and continuous medical treatment will be necessary. The eye treatment required consists of copious lavage with warm saline repeated not less frequently than at hourly intervals. The rapidity with which such treatment can be carried out after eye contamination is of great importance; serious injury will in no case be averted, but permanent blindness may be prevented by prompt and thorough but gentle treatment.

67. Paralysant Gases: Diagnosis

Hydrocyanic acid gas and hydrogen sulphide gas are commonly known as paralysant gases. This description refers to their action in very high concentrations, when, by a paralysis of the respiratory centre in the brain, they quickly produce sudden unconsciousness and failure of respiration. In weak concentrations they are not harmless, but in comparison with other gases their action is of a mild character.

The recognition of casualties caused by these gases rests on a history of exposure to an atmosphere with a smell either of bitter almonds or rotten eggs; this being followed by rapid loss of consciousness, with or without convulsions, and by failure of respiration. Subsequent recovery is usually rapid and without complications.

68. Carbon Monoxide: Diagnosis

The recognition by a doctor of cases of carbon monoxide poisoning in war time should not be difficult if he is familiar with the possible sources of such poisoning (as detailed in Section 47) and if he can obtain a history of the circumstances under which the condition occurred.

If unconsciousness has resulted, or even if breathing is failing, the treatment is immediate removal from the poisonous atmosphere and recourse to artificial respiration in pure air. After this the differential diagnosis should be considered.

The distinction of severe carbon monoxide poisoning from other comatose states rests chiefly on the history of the case, the pinkness of the lips in the early stages, and the examination of the blood by the test given in Section 47. Less severe degrees of poisoning occur with symptoms of headache, giddiness, breathlessness on exertion, weakness in the legs, and cerebral states varying widely from irritability to mental confusion. Such cases do not call for artificial respiration, but they need careful control lest over-exertion harm a heart which is already strained by anoxaemia.

69. Nitrous Fumes: Diagnosis

The recognition of cases of poisoning by these gases presents many difficulties in the early stages owing to the insidious nature of the onset. The incomplete

detonation of a charge in a mining gallery, or the breathing of the vapour arising from the spilling of nitric acid on a wooden floor, may evoke a little coughing which soon subsides: hours later, however, an oedema of the lungs may follow and prove fatal.

The recognition of such potential casualties must to a large extent depend on the history of exposure; when such a history is establishd, the treatment must, as in cases of phosgene gassing, be largely that of watchful anticipation for a period of at least 24 hours. In the meantime, complete rest is imperative, and if pulmonary oedema should develop, its recognition and treatment follow closely the lines already laid down for cases of phosgene gassing.

70. Screening Smokes: Diagnosis

These smokes are harmless in the concentrations which form effective screens, and casualties do not therefore occur except in the case of men who may come in contact with the compounds before they have been dissipated in the form of smoke.

TABLE OF GASES

Example.	Properties.	Effects.	Remarks.
Tear Gases—			
Chlor-aceto-phenone (C.A.P.) (*non-persistent.*)	A solid. Almost invisible in gaseous state. Recognised by irritation to eyes.	Irritation or stinging of eyes followed by copious flow of tears and spasm of eyelids. Slight skin irritation.	Respirator affords complete protection.
Ethyl-iodo-acetate (K.S.K.) (*persistent*).	A dark brown liquid. Invisible in gaseous state. Recognised by irritation to eyes.	Do., but no skin irritation.	Do.
Bromo-benzyl-cyanide. (B.B.C.) (*persistent*).	A yellowish-brown crystalline solid when pure. As used—a liquid mixture. Invisible in gaseous state.	Similar to K.S.K.	Do.
Nose Irritant Gases—			
Di-phenyl-chlor-arsine (D.A.) (*non-persistent*).	A colourless, crystalline solid which when heated gives off an almost odourless smoke. Generally invisible except near the source. Can still be effective although not visible.	Produces sneezing ; burning pain in chest, throat, nose and mouth ; mental depression.	Do.

Example.	Properties.	Effects.	Remarks.
Di-phenyl-chlor-arsine (D.A.) —*contd.*	Recognised by effects, which are slightly delayed.		
Di-phenyl-amine-chlor-arsine (D.M.) (*non-persistent*).	A bright yellow, crystalline solid. General properties similar to D.A.	Produces sneezing; burning pain in chest, throat, nose and mouth; mental depression.	Respirator affords complete protection.
Di-phenyl-cyano-arsine (D.C.) (*non-persistent*).	A crystalline solid with properties similar to D.A.	Do.	Do.
Lung Irritant Gases—			
Chlorine (*non-persistent*).	A gas—greenish colour. Corrodes metals. Is dissolved in water. Will eventually rot clothing. Smell of bleaching powder.	Highly lethal, owing to damage to lungs. Early symptoms: cough and watering of eyes. Signs of lung damage develop later.	Do.
Phosgene (*non-persistent*).	A gas—almost invisible. Corrodes metals. Is rendered less effective by heavy rain. Smell of musty hay	Do.	Do.

Di-phosgene (*non-persistent*).	A colourless liquid. Invisible in gaseous state.	Do.	Do.
Chloropicrin (P.S.) (*semi-persistent*)	A colourless, volatile liquid with a pungent odour.	Generally similar to above. Also causes sickness and slight irritation of eyes.	Do.
Blister Gases—			
Mustard gas (H.S.) (*very persistent*).	An oily liquid which may vary in colour from dark brown to straw yellow. Soluble in oil and spirits. Neutralised by bleaching powder. Great power of penetration. Smell of garlic, onions, horse-radish or mustard. Liquid may be seen.	(i) *Vapour.* (*a*) In eyes; irritation and inflammation with swelling and temporary loss of vision usually develop within 24 hours, but tears earlier if the vapour concentration is high. (*b*) In lungs; loss of voice and cough. Later possibly bronchitis and bronchopneumonia. (*c*) On skin; redness, irritation and perhaps blisters; but to an unprotected man, the eye damage is worse than the lung or skin effects. (ii) *Liquid.* (*a*) In eyes; immediate irritation, eye closes in about one hour.	Respirator protects eyes and lungs only.

APPENDIX A—*contd.*

Example.	Properties.	Effects.	Remarks.
Mustard gas (H.S.)—*contd.*		(ii) *Liquid*—contd. (*b*) On skin ; no irritation, redness in 2 hours followed by blister in 12 to 24 hours. (*c*) The effect of swallowing food contaminated by liquid mustard gas is severe injury to stomach and intestines.	
Lewisite (*very persistent, but less so than mustard gas*).	A colourless liquid when pure ; brown in the crude state. Gives off an invisible gas. Is rapidly destroyed by water and any alkali. Great power of penetration. Has a low freezing point. Smell of geraniums.	(*i*) *Vapour.* Causes severe irritation to nose. Hence respirator will be adjusted immediately so that there will be no permanent effects on eyes, nose or lungs. Is less effective on skin than mustard gas vapour. (ii) *Liquid.* (*a*) In eyes ; immediate effect and permanent injury. (*b*) On skin ; blisters develop more rapidly than with mustard gas.	Respirator protects eyes and lungs only.

Paralysant Gases—			
Hydrocyanic acid (*non-persistent*).	A colourless, volatile liquid with a smell of bitter almonds.	Small amounts of vapour cause giddiness and headache ; larger doses cause unconsciousness and death.	Respirator affords complete protection.
Hydrogen sulphide. (*non-persistent*).	A colourless gas with characteristic smell of bad eggs.	Small doses cause irritation of eyes and nose ; large doses cause unconsciousness and death.	Do.
Other Gases—			
Carbon monoxide. (*non-persistent*).	A colourless, odourless gas.	Causes giddiness, tiredness, then unconsciousness and death.	Respirator does *not* protect against this gas. Unlikely to be used as a war gas on account of its physical properties.
Nitrous fumes (*non-persistent*).	Reddish-brown fumes. Pungent smell.	Irritation of nose, throat and lungs.	Respirator gives adequate protection for limited periods.

FORMULAE AND PHYSICAL CONSTANTS OF GASES

Substance.	Chemical Formula.	Boiling Point.	Melting or Freezing Point.
Tear Gases.			
Chlor-aceto-phenone (C.A.P.)	$C_6H_5.CO.CH_2Cl$	245°C. (473°F.)	54° to 59°C. (129·2° to 138·2° F.)
Ethyl-iodo-acetate (K.S.K.)	$CH_2I.CO.O.C_2H_5$	180°C. (356°F.)	Pure: — 21°C. (— 5·8°F.) K.S.K.: — 27°C. (— 16·6°F.)
Bromo-benzyl-cyanide (B.B.C.)	$C_6H_5.CHBrCN$	242°C. (467·6°F.)	24·8°C. (76·6°F.)
Nose Irritant Gases.			
Di-phenyl-chlor-arsine (D.A.)	$(C_6H_5)_2AsCl$	333°C. (631·4°F.)	38° to 40°C. (100·4° to 104°F.)
Di-phenyl-amine-chlor-arsine (D.M.)	$(C_6H_4)_2NHAsCl$	410°C. (770°F.)	195°C. (473°F.)
Di-phenyl-cyano-arsine (D.C.)	$(C_6H_5)_2AsCN$	346°C. (654·8°F.)	33°C. (91·4°F.)

Lung Irritant Gases.			
Chlorine	Cl_2	—33·6°C. (—28·6°F.)	— 102°C. (— 151·6°F.
Phosgene	$COCl_2$	8·2°C. (46·8°F.)	— 118°C. (— 180·4°F.
Di-phosgene	$Cl.COOCCl_3$	128°C. (262·4°F.)	— 57°C. (— 70·6°F.))
Chloropicrin	CCl_3NO_2	112°C. (233·6°F.)	— 69°C. (— 92·2°F.))
Blister Gases.			
Mustard gas (H.S.) ($\beta\beta$ di-chloro-di-ethyl sulphide)	$(CH_2Cl.CH_2)_2S$	217°C. (422·6°F.)	Pure: 14·4°C.(57·9°F.) Crude: 6°C. (42·8°F.)
Lewisite (β chloro-vinyl-di-chlor-arsine)	$(CHCl:CH)AsCl_2$	190°C. (374°F.)	— 13°C. (8·6°F.)
Paralysant Gases.			
Hydrocyanic acid (prussic acid)	HCN	26°C. (78·8°F.)	— 14°C. (6·8°F.)
Hydrogen sulphide (sulphuretted hydrogen)	H_2S	— 62°C. (— 79·6°F.)	— 86°C. (—122·8°F.)
Other Gases.			
Carbon monoxide	CO	— 190°C. (— 310°F.)	— 207°C. (— 340·6°F.)
Nitrous fumes	NO, NO_2, N_2O_4, etc.	NO_2:26°C. (78·8°F.)	NO_2: — 10°C. (14°F.)

§ 22.

APPENDIX C

ROUTINE OF OXYGEN ADMINISTRATION

The cases requiring oxygen treatment should be aggregated in wards set apart and specially equipped for their reception.

The cylinders* of oxygen, one for each bed with a 50 per cent. reserve, are best grouped as a battery of cylinders. This grouping of cylinders should be arranged close to, but outside, the actual ward, in a lobby or passage. This permits of cylinder replacement being carried out by an untrained orderly or porter without his actually entering the ward. Every cylinder should have its pressure gauge and the orderly can very soon be trained to watch these gauges, to detach individual cylinders as and when the gauge shows it to be necessary, and to replace it by a fresh cylinder from the immediately available reserve. He can also be responsible for replenishing the local reserve from the hospital general store at definite times arranged with the hospital quartermaster or storekeeper. All this can be done without any interference with ward routine if the cylinder group is outside the ward.

The cylinders should be connected to a main storage and break-pressure tank by means of individual pipes leading to a manifold. From this tank one main pipe should lead down each side of the ward, for the full length. These pipes may be connected at the other end to form one circuit. There should be a T-piece over each bed to provide a separate supply for each patient. Each of these bed-leads should have a Haldane administration apparatus, or some modification of that principle, complete with graduated flow control and administration mask or tent.

For the continuous and long-continued administration which these cases demand this centralised layout is more satisfactory, more economical of nurse-power,

* Contents of cylinder, 20 cub. ft. = 560 litres.

and easier to control than a bed-by-bed layout. The fittings can be made of lead or gas piping, and can be fixed permanently in position. A part, if not the whole, of the individual bed-leads must be of rubber tubing.

When the layout is complete the cylinder valves can be opened and each separate administration unit tested for a satisfactory oxygen flow.

Administration can best be carried out by a Haldane apparatus (or some modification of it), but if not available it can be done by the nasal catheter or double nasal catheter.

The great advantage of the special apparatus is that the flow is mechanically controlled, and the actual quantity being given is shown on the graduated control tap. Moreover, wastage during expiration is avoided by the system of valves and collecting bag which is part of the apparatus. These two factors combine to reduce avoidable waste of oxygen. This is a most important matter if many cases have to be treated over long periods since under war conditions there may be serious difficulties of supply and distribution.

The ward is now equipped and ready to receive patients, and routine treatment may begin. This routine, as applicable to any one patient, will now be followed through.

Whenever possible, oxygen administration should be in the hands of nurses specially detailed and coached for this work, the whole of the general nursing being carried out by other members of the nursing staff.

It is best if one nurse, doing oxygen administration routine, be given five beds. These beds must all be in one ward and running in sequence, not dotted about.

The nurse must have a time sheet or note book, and each bed must have a bed-head writing pad or, better still, a slate.

At the time of beginning oxygen administration to any given patient the nurse makes two entries: in her note book, the time to the nearest minute, *but adding 25 minutes;* on the patient's bed-head pad or slate, a short note of the clinical condition in simple terms under three heads, (1) general demeanour of case, (2) colour, (3) condition of pulse.

It is well to begin every administration at a flow rate of 3 litres per minute. Almost immediate clinical improvement, especially of the colour, is the criterion of whether the flow is rapid enough. In the absence of this expected improvement, it is permissible to increase the flow, but it is no use increasing it beyond 10 litres per minute. Any more rapid flow will merely result in avoidable wastage without any advantage being gained. If the hoped for clinical improvement is not evidenced with this maximal permissible flow of 10 litres per minute it means that the case is all but hopeless. In a few cases the flow can be cut down below 3 litres per minute.

The rate of flow having been adjusted and some clinical improvement being established, this patient ceases to be the responsibility of the nurse in charge of oxygen *for the next 25 minutes.* During this period it is the responsibility of the other members of the nursing staff to see that the administration is as uninterrupted as is humanly possible.

At the end of the 25 minute period, marked for her on her time sheet by having added 25 to the original time of starting oxygen, she returns to this particular bed.

She alters the bed-head notes as required by the new condition of the patient. She turns off the oxygen and the administration is discontinued for five minutes (this period also is noted on the time sheet, and worked to by the nurse, in minute periods). At the end of

the five minutes, or sooner if the clinical signs evidence any retrogression, the administration is restarted for another 25 minutes.

This routine of 25 minutes on, five minutes off, 25 minutes on, five minutes off, is continued *until the five minutes' interval produces no clinical setback.* If and when this stage is reached, it is usual to find that a turning point has been reached, no further oxygen is needed, and recovery is certain unless there should be a relapse due to some unexpected sequela such as pneumonia.

The danger which is being fought is the very serious one of oxygen starvation, against which the patient cannot stand up for very long.

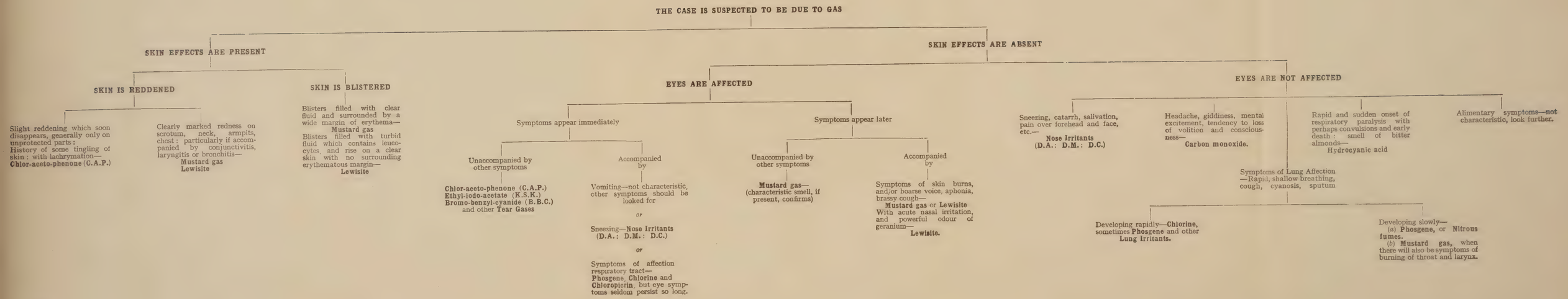
APPENDIX D

OUTLINE DIAGNOSIS CHART FOR CASES DUE TO GAS

See Table opposite

APPENDIX D

OUTLINE DIAGNOSIS CHART FOR CASES DUE TO GAS



INDEX

(40215—34) Wt 3150—817 25,000 7/37 P. St. G. 373

Official Publications on Air Raid Precautions

HANDBOOKS

See list on page ii within.

MEMORANDA

No. 1 Organisation of Air Raid Casualties Services (*2nd edition*) 6*d*. (7*d*.)

No. 2 Rescue Parties and Clearance of Débris (*2nd edition*) 2*d*. (2½*d*.)

No. 3 Organisation of Decontamination Services (*1st edition*) 2*d*. (2½*d*.)

No. 4 Air Raid Wardens (*1st edition*) 2*d*. (2½*d*.)

Prices are net, those in brackets include postage.

H.M. STATIONERY OFFICE

LONDON, W.C.2	Adastral House, Kingsway
EDINBURGH 2	120 George Street
MANCHESTER 1	26 York Street
CARDIFF	1 St. Andrew's Crescent
BELFAST	80 Chichester Street

or through any bookseller

S.O. Code No. 34-229-3